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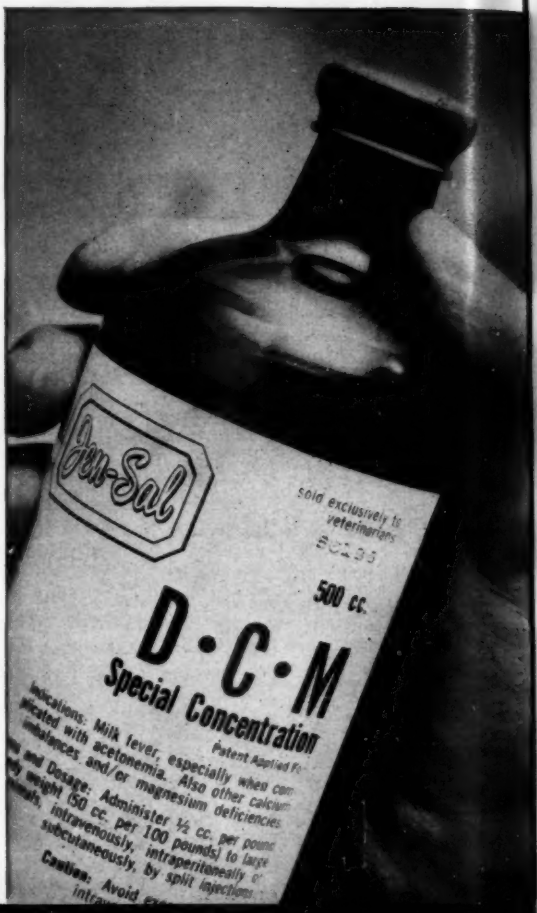

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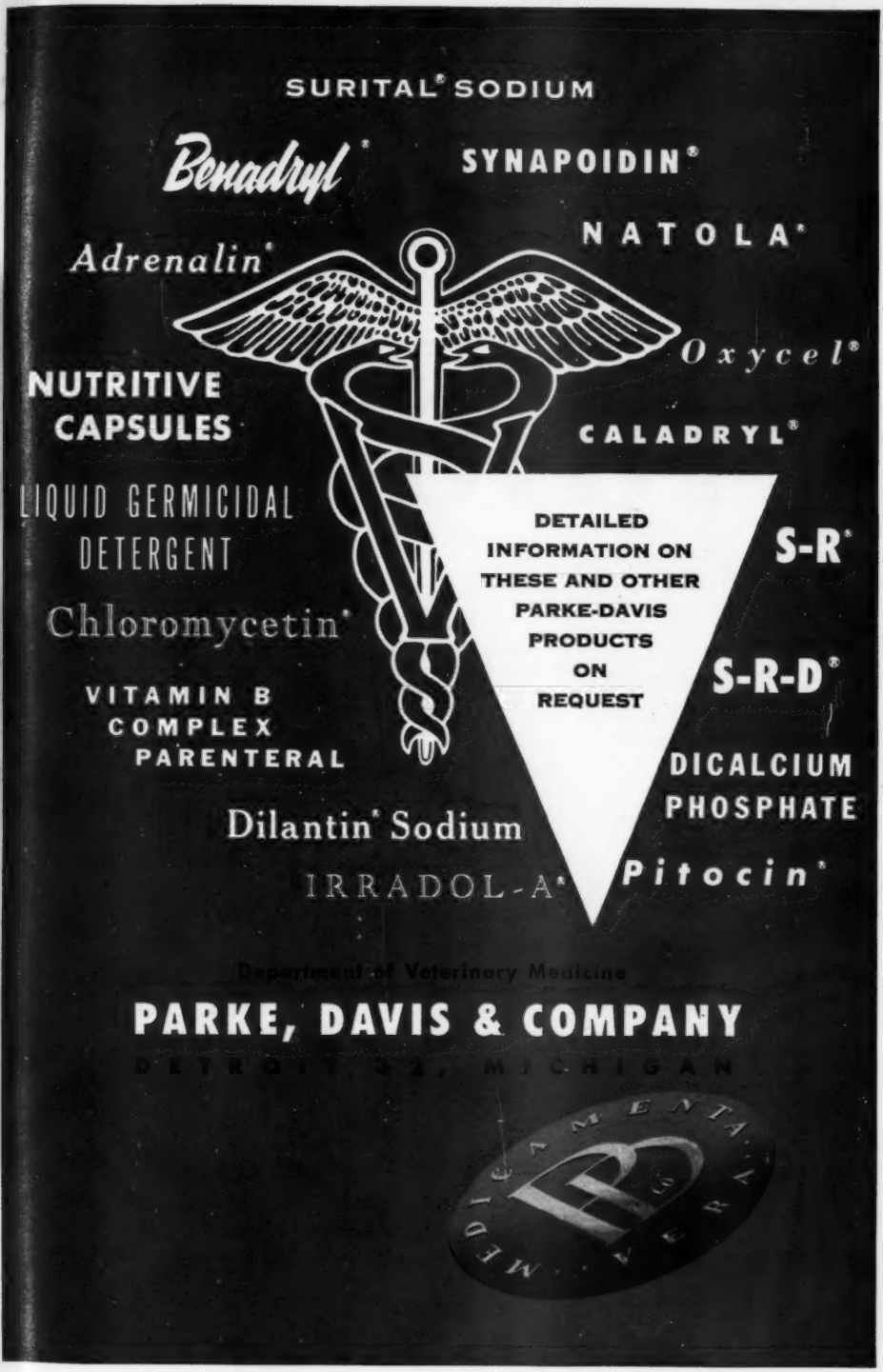
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Sex Hormones In Veterinary Practice†

A. H. CRAIGE, JR., V.M.D., Indianapolis, Indiana

New knowledge has stripped many of the old mysteries from the action and interaction of secretions of ductless glands (hormones) on physiological manifestations, particularly as they govern the sexual cycle in females. They are now regarded in their true light, as definite chemical substances which can be synthesized economically and which elicit reproducible physiological reactions that can be observed clinically. As more knowledge accumulates on the pharmacologic properties of these hormones and their interrelationships, veterinarians can expect better results and an ever expanding demand for their use.

THE role of sex hormones in regulating body functions has been recognized since the dawn of medicine. The ancients practiced castration, on both animals and man, in order to prevent natural body changes of maturity. More recently surgeons began experimenting in the opposite direction, gonadal implants to supplement natural deficiencies in sex hormones. But rapid progress in this field began about 20 years ago with the chemical isolation of the sex hormones in pure form, and their synthesis at relatively low cost.

Since then pure sex hormones (testosterone, progesterone, and the synthetic estrogen, diethylstilbestrol) have been under intensive study in both laboratory and clinic. As a result, a wealth of information has accumulated, shedding light on the complex physiology of sex. While this knowledge still is incomplete, and many fundamental questions remain to be answered, there is enough to point the way to useful application of these hormones in veterinary medicine. In fact, they are now in wide use in an ever increasing number of clinical problems. The purpose of this brief review is to summarize the theoretical background for these uses, to the end that their clinical possibilities can be realized more fully.

Methods of Administration

One of the problems in using sex hormones in veterinary practice has been that of establishing and maintaining effective blood levels with doses at intervals long enough to be practical. Pellets implanted surgically provide prolonged action, to be sure, but their action is of a low blood level and difficult to regulate. Oil solutions, on the other hand, produce blood levels which can be varied to suit the needs, but they are transient. In order to accomplish the prolonged stimulation so often required, it is necessary to inject an oil solution every day or so. Obviously this is impractical in most cases veterinarians are called upon to treat.

The need for long, sustained action, at effective hormonal levels, led to the development of a special vehicle (reposital®) in which these hormones are in solution, but from which they quickly precipitate in the aqueous medium of body tissues. As soon as reposital solutions are injected into muscle tissue, the hormone precipitates and is deposited as millions of tiny tissue implants from which they feed the hormone continuously into the blood stream. As a result of the many particles implanted,

†Presented at the 36th annual meeting, Southern Veterinary Medical Association, November 9-11, 1953, Atlanta, Ga.

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high blood hormone levels develop promptly and persist for long periods. These prolonged physiologic levels enable the veterinarian to apply effective, continuous hormone therapy with injections, often weeks apart.

Female Sex Hormone Functions

Peripheral and Central Effects. The hormones produced by the ovary, estrogen and progesterone, act upon many organs of the body. These actions may be classed as peripheral effects and are of particular interest in therapeutic use of hormones. They include effects on the uterus, external sex glands, mammae, muscle development, hair growth, and many others.

At the same time these hormones are working peripherally on body tissues, they are also working centrally on the anterior pituitary gland, which regulates the natural production of these same hormones by the ovary. Since these central effects often result in suppressing the ovarian output of the very hormone being administered, they account for many of the confusing, and apparently contradictory, symptoms often observed in practice.

These central effects are taken up spearately. Let us consider now the peripheral effects of these hormones—their end-results on the body. The most important are concerned with the uterus, the mammary glands, and estrum. In general, the peripheral effects of estrogens have to do with preparing the body for pregnancy, those of progesterone with maintaining pregnancy.

Uterus. Estrogen induces changes in the endometrium which increase its resistance to infection. Thus, diethylstilbestrol may be said to "toughen" the endometrium. This is the basis for its use in pyometra and other uterine infections. It is likewise the basis for its use in mismating, for toughening also makes the endometrium less hospitable to the implanted embryo, and can cause a fetus to be thrown off, just like an infection.

Progesterone has the opposite effect. It "softens" the endometrium, making it more hospitable to the fertilized ovum. For this reason a dose of progesterone given to the "repeat breeder" at the time of insemination can result in conception and a live calf. The same therapy, for much the same reason, can stabilize the uterus of the habitual mid-term aborter.

These hormones also act on the uterine musculature. Diethylstilbestrol increases uterine tone, for which it is used therapeutically in weak labor, mummified fetus, and retained placenta. Undoubtedly the endometrial toughening effect referred to above also is important in the latter conditions, particularly when infection is present, but the uterine contractions induced by diethylstilbestrol serve an important function whenever the uterus must be emptied.

In addition to emptying the uterus, diethylstilbestrol causes the cervix to dilate. This action can be useful in prolonged gestation, hydrops amnii, and other clinical problems characterized by cervical spasm. This is an action physiologically comparable to uterine contraction, for the same stimuli that contract the uterus cause the cervix to relax.

Progesterone inhibits uterine contractions, for which it has been found useful to prevent miscarriage when shipping and handling cows in late term, when they are heavy with calf. It is also the basis for its use to correct vaginal prolapse. Moreover, it is highly probable that this relaxing effect on the uterus functions in conjunction with the aforementioned endometrial action to prevent mid-term abortion. It is generally regarded, however, that the endometrial effects are more important during early pregnancy, and the muscular effects late in term. In all these peripheral actions on the uterus, progesterone and estrogen are antagonistic, both on the endometrium and the uterine muscle.

Mammary Gland. In the development of the mammary gland, estrogen stimulates growth of tubules and mammary duct system, whereas progesterone is responsible for lobulo-alveolar proliferation. As a result of the alternate bombardment by these hormones with each sexual cycle, the mammae acquire size and productive capacity. But it is the high concentration of progesterone during pregnancy that finally prepares the gland for active milk secretion.

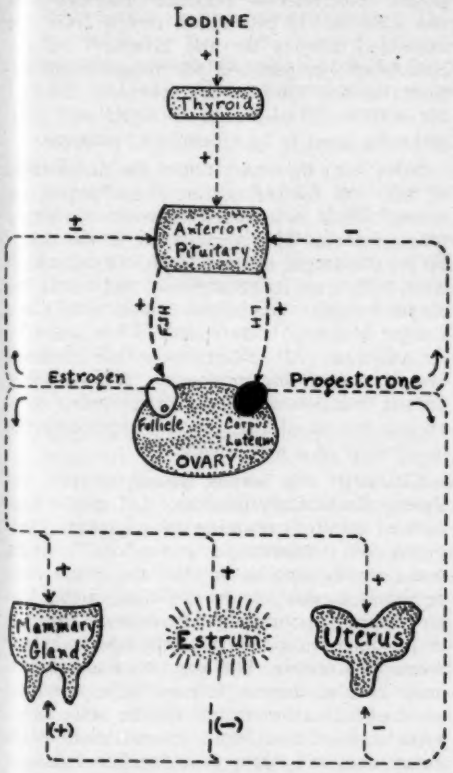
A third hormone appears to be necessary to initiate milk flow, after estrogen and progesterone work together to prepare the gland. That "trigger" hormone is prolactin, secreted by the anterior pituitary gland. Progesterone is believed to inhibit the release of prolactin, and block its action in the mammary gland. Accordingly, it is not until the very end of term, when blood progesterone level falls abruptly, that prolactin takes hold and lactation starts.

This alternate stimulation of the mammary gland by estrogen and progesterone has been applied experimentally to initiate milk flow in virgin heifers and barren cows. Results have been encouraging, but not always followed by a gratifying flow of milk. These discrepancies may be attributed to insufficient preparation

of the glands, or overdosage, particularly with estrogen. They may be due to deficiency in circulating prolactin, from the effects of progesterone.

According to one experimental procedure, doses of diethylstilbestrol and progesterone are followed after a few weeks by a dose of the estrogen alone. Used this way, it is important to keep the dose of diethylstilbestrol down low enough to avoid severe mammary engorgement, which would interfere with milk flow. When it is necessary to use excessive doses for other peripheral actions (*e.g.* on the uterus), the severe mammary congestion which may occur as a side action usually can be counteracted by a dose of progesterone.

DIAGRAM. Regulation of the Female Sex Hormones



While the ovary occupies the central role in this drama, iodine takes the lead. Iodine stimulates (+) the thyroid to secrete thyroxin, which then stimulates the anterior pituitary to secrete its gonadotrophins, alternately FSH (follicle stimulating hormone) and LH (luteinizing hormone). Now in turn, FSH causes ovarian follicles to form and secrete estrogen, and LH causes follicles to be replaced with corpora lutea, which secrete progesterone.

The estrogen released by the follicle produces peripheral stimulant effects (+) on the mammary gland, uterus and other sex organs, and central effects of the pituitary, consisting of both stimulation (+) and depression (−). In contrast, the progesterone secreted by the corpus luteum both supplements and counteracts the peripheral actions of estrogen. Centrally, progesterone only inhibits (−) the pituitary, the basis for its clinical use in cystic ovaries, and to delay estrum.

Estrum. The complex physiologic changes known as estrum are produced by the estrogens and suppressed by progesterone. For most clinical purposes estrum is regarded as an undesirable side action, to be suppressed by a neutralizing dose of progestrone as soon as the main purpose of the estrogen is accomplished. For example, to prevent pregnancy after mismating, it is prudent to give a large enough dose of diethylstilbestrol to be sure the patient cannot conceive (0.5-1.0 mg. per lb.). Since this dose may cause prolonged estrum, directions should be given the owner to expect this side effect, and if it is bothersome, to request a return visit a week later. At this time the patient may be given a quieting dose of progesterone (1 mg. per lb.).

One clinical condition in which estrogen is used to produce estrum is illustrated by the cow with so-called "silent heat." Evidently these animals ovulate normally, but insufficient estrogen is produced to cause visible estrum, and the owner does not know when to breed the cow. Small doses of diethylstilbestrol are given to raise the "basal estrogen level," so that when the next follicle matures the cow is visibly in heat and can be bred at the right time. Overdose must be avoided, for it is just as much of a problem for the cow to be continuously in heat as never in heat.

Many more times owners find it expedient to prevent females from coming in heat at their natural period. Progesterone has found wide favor to prevent this inconvenience in hunting bitches, racing greyhounds, show dogs, and other females where the occurrence of estrum would interfere with their usefulness. To some degree this action of progesterone appears to be peripheral, for it has limited value for the animal in heat. Most of its effect must be central, however, suppressing the development of the ovarian follicle, since for best results, progesterone must be given before there are visible symptoms of estrum.

Other Applications. A few other clinical uses have been reported for the female sex hormones which might be classified as due to peripheral actions. Most noteworthy is the widely accepted application of diethylstilbestrol

in urinary incontinence of spayed bitches. While the mechanism is not clearly understood, it has been attributed to a tonic effect of the estrogen on the sphincter of the bladder, comparable to its action on the uterine wall.

Most other clinical applications commonly employed in veterinary medicine are ascribed to the central effects of these hormones, exerted through action on the anterior pituitary gland. These effects become clear only against a background of the normal regulation of the ovary, as it is controlled by the pituitary. Before attempting to explain these indirect actions, let us review briefly the mechanics of normal sex hormone regulation in the female.

Female Hormone Regulation

The story of sex begins with the anterior pituitary gland, where many endocrine functions originate. The anterior pituitary secretes two gonadotrophic hormones, follicle-stimulating hormone (FSH) and luteinizing hormone (LH). The gonadotrophic hormones stimulate the ovary to produce the gonadal hormones, estrogen and progesterone. The pituitary, in turn, is subject to regulation by both these sex hormones and also by thyroxin, secreted by the thyroid gland. Thus, it is convenient to consider the endocrine control of sex at three levels: (1) Pituitary (FSH, LH); (2) gonadal (estrogen, progesterone); and (3) thyroid (thyroxin).

The gonadotrophic hormones, FSH and LH, have leading roles in the drama of sex, because they regulate the production of sex hormones by the gonads. It is the sex hormones themselves, however, that produce the body changes characteristic of sexual development, as described in the preceding paragraphs. Both these gonadotrophins are secreted more or less continuously, but alternately either FSH or LH predominates. It is this rhythmic domination of one gonadotrophin, then the other, that produces the normal female sexual cycle.

Female Cycle. Follicle stimulation, which predominates when FSH is in power, causes a follicle to start developing in the ovary. That is as far as FSH takes it. Pituitary LH must then take over to complete maturity of the follicle and cause it to rupture and discharge the ovum (ovulation). When FSH persists in power, or LH is slow to take over, excessive follicle growth results in cystic ovaries.

This FSH phase of the female sex cycle is characterized by the body changes and physiologic reactions described as "estrogenic." At this time the developing follicle is throwing out more and more estrogen, causing the same peripheral effects as a dose of diethylstilbestrol. As mentioned before, they all have to do with preparing the body for pregnancy and culminate in estrum and coitus.

At this climax in the estrogenic stage, the pituitary suddenly shifts production to LH and the ovary now enters the "progesterogenic" stage. Experimental evidence indicates that this shift to LH production results from the combined actions on the pituitary by accumulated estrogen and the progesterone secreted at this time by the granulosa cells of the follicle. Thus, both estrogen and progesterone seem to be needed for ovulation.

Now, with the ovary under the domination of LH, the follicle ruptures, discharging its ovum, which is intended to meet a waiting spermatozoon. The opening left in the ovary by the discharged ovum fills with blood, which clots, soon loses its hemoglobin, and forms the corpus luteum. The corpus luteum now takes charge of many body functions by secreting progesterone. All efforts now are directed toward maintaining pregnancy. Among other actions mentioned before, progesterone suppresses release of LH, thereby preventing the maturation of a new follicle.

Ultimately the corpus luteum recedes, releasing the pituitary to secrete LH, and a new follicle matures, resulting in ovulation. Implants of progesterone (repositol) can be made at this time to simulate the effect of a persistent corpus luteum and thus further delay follicle maturation. Persistence of the corpus luteum depends normally upon whether pregnancy occurs, but premature disappearance of the corpus luteum is a common cause of habitual abortion. On the other hand, persistence of the corpus luteum when pregnancy does not occur is a familiar cause of failure of cows to come in heat. Thus cystic ovaries and retained corpus luteum may be regarded as comparable interruptions in gonadotrophic rhythm, but occurring at different stages in the ovarian cycle.

Reaction on the Pituitary. Some of the most important therapeutic effects of sex hormones are best explained as reactions on the gonadotrophic secretion of the anterior pituitary gland. These are the "central" effects of the sex hormones. Apparently the anterior pituitary secretes FSH spontaneously and continuously, but as follicles ripen, the estrogen they secrete reacts back upon the pituitary to (1) suppress the release of FSH, and (2) stimulate the release of LH. These effects result

normally in ovulation, followed by formation of the corpus luteum. This stimulant effect of estrogen on the pituitary LH constitutes a basis for the repeated administration of small doses of diethylstilbestrol to mature an infantile ovary.

Following formation of the corpus luteum, the progesterone it secretes suppresses release of both FSH and LH, thereby preventing the development and maturation of new follicles as long as the corpus luteum persists. This is the basis for the administration of progesterone to prevent nonpregnant females from coming in heat. Comparatively high doses of progesterone are needed to suppress estrum.

Evidently higher blood progesterone concentrations are required to suppress FSH than to suppress LH. According to this concept, cystic ovaries result when the blood level of progesterone is only high enough to suppress LH, but not high enough to suppress FSH. At this intermediate progesterone level ovarian follicles are stimulated to enlarge, yet in the absence of LH they cannot ripen and ovulate. Thus, follicle overgrowth occurs. The obvious solution is to inject more progesterone, which has been done with excellent results.

Persistence of the corpus luteum is necessary to maintain pregnancy, although the length of time required varies with species and individuals. Its removal from a cow before the 200th day of pregnancy usually leads to abortion. This protective action is attributed to the muscle relaxation and endometrial vascularity maintained by the progesterone it secretes. Normally, when the corpus luteum disappears, its protective function is taken over by the placenta. A gap between luteal regression and placental response is regarded as the common cause of habitual abortion. Progesterone replacement therapy has proved highly effective in bridging that gap and preventing abortion.

Male

Pituitary regulation of the testicle is comparable to that of the ovary. In the male, FSH stimulates the seminiferous tubules to produce spermatozoa, whereas LH causes the interstitial testicular cells to secrete the male sex hormone, testosterone. In addition, the supporting cells of the tubules, Sertoli cells have endocrinologic interest, because they secrete estrogen. When tumor growth involves these Sertoli cells, as is not uncommon in the dog, it produces a characteristic clinical picture of feminization and bilateral alopecia.

Testosterone has two distinct functions. It stimulates the body tissues to develop along the lines of the mature male (conformation, testicular descent, growth of male genitalia) and induces libido, or responsiveness to female estrum. These are functions which serve as the basis for using testosterone therapeutically in a wide range of physical and sexually functional disturbances in males. For androgenic replacement it is used to enable the breeder to get a few more services out of a senile bull, to cause the testicles of a young ridgling to descend, to dilate the urethras of steers, castrated tomcats, and wethers when they are in danger of obstruction by calculi, and to counteract muscle weakness of castrates.

Centrally, testosterone suppresses pituitary production of both FSH and LH. This is the basis for its clinical use to counteract estrogenic symptoms. Its capacity to inhibit lactation, and to reduce the size of mammary tumors, is attributed to this central action. Other testosterone uses sometimes ascribed to gonadotrophic suppression include the correction of bilateral alopecia in cases of Sertoli cell tumor, and false pregnancy in bitches.

Apart from these androgenic functions, testosterone is also a metabolic stimulant. Lazy, fat old dogs, with dainty appetites, often show marked improvement after testosterone. They become more active, appetites improve, yet they lose weight. In these cases, testosterone stimulates smouldering tissue metabolism, causes increased nitrogen retention, and corrects anemia. This rejuvenation works equally in overaged females and in males. The same principle is applied to the use of testosterone to restore muscle vigor in wasting diseases and to stimulate weak and premature young animals. In this sense testosterone can be regarded as a metabolic tonic rather than an androgenic.

Role of the Thyroid

The thyroid gland occupies a dominating position in reproduction. Through a stimulant action of its hormone, thyroxin, on the pituitary, the thyroid determines the output of gonadotrophins, the regularity of ovulation, and the production by the ovary of estrogen and progesterone. Thyroid deficiency is characterized by failure to ovulate normally. This causes breeding irregularities which can lead to complete reproductive failure. Cattle from which the thyroids have been removed fail to show physical signs of estrum.

In studies on the natural yearly cycle of estrum and ovulation in sheep, it has been

shown that thyroid secretion is four times greater in the winter breeding season than during the sexually inactive summer. Since the condition of the ovaries and pituitary are the same in both seasons, it is difficult to escape the conclusion that the thyroid plays the major role in regulating reproduction.

Iodine Stimulation. Iodine long has enjoyed a reputation in reproductive disturbances. It has been reported beneficial in such widely divergent conditions as cystic ovary, retained corpus luteum, infantile ovary, weak estrum of animals, amenorrhea in humans, male impotence, habitual abortion, and many others. While many of these reports must be viewed with restraint, most clinicians who have used iodine extensively are convinced that it has merit.

Since an adequate intake of iodine is essential for the normal secretion of thyroxin, it is not difficult to understand how iodine could benefit animals with frank deficiency. But in the case of animals without apparent iodine deficiency, we do not know to what extent these reproductive problems may be due to a mild, unsuspected degree of iodine deficiency.

The effectiveness of iodine therapy in reproduction problems was the subject of recent report (Baker, 1953) in which an organic iodine was administered routinely to more than 700 "shy breeders." These were cows that had not settled on previous services, but otherwise appeared normal. The treatment consisted in 1 oz. of HI-amine compound® added to the grain ration morning and night, beginning eight days before the next anticipated breeding date. This treatment was followed by conception in 90% of the cows with the next service.

Summary

The sex hormones (estrogen, progesterone, testosterone) are produced by the sex glands in response to the gonadotrophins (FHS and LH) first produced by the anterior pituitary gland. Thus, the sex hormones may be regarded as the "effector" agents, while the gonadotrophins are their "regulators." These "effector" agents are used in veterinary medicine for two distinct types of physiologic action: peripheral effects, such as are exerted directly on the body organs, like the uterus, mammary glands, external sex organs; and central actions, which revert back to the pituitary, and in that way affect the gonads. Progesterone, for example, works peripherally on the uterus to prevent abortion, and centrally through the pituitary, where it suppresses FSH, to delay estrum.

Many clinical uses have been developed for the sex hormones, both in females and males. Naturally, most of the estrogen and progesterone is used in females, and the testosterone in males. But by no means are the hormonal actions confined to their sex of origin. Testosterone stimulates metabolism in overaged females as well as males, and estrogen counteracts prostatic enlargement by suppressing release of gonadotrophins by the pituitary.

Newer knowledge has stripped much of the old mystery from sex hormones. Now they are regarded in their true light, as definite chemical substances which elicit reproducible reactions in the body like atropine, digitalis, and other drugs. As more knowledge accumulates on the pharmacologic properties of these hormones, and better understanding of the physiologic processes in the cases where they are used, veterinarians can expect still better results and an ever expanding demand for their use.

®Ethylenediamine dihydriodide, 20 gr. per oz. in sodium chloride—Pitman-Moore Co., Indianapolis, Ind.

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(Continued on page 190)

Edema Disease In Swine

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ALTHOUGH edema disease apparently is not of great importance in the overall swine disease picture, at times it can be of major concern to the individual veterinarian when he is confronted with an outbreak within a short time after vaccinating a drove for hog cholera. When losses are heavy, the swine owner is almost certain to believe that the vaccination played a part in the disease picture. Even though there are good reasons for believing that vaccination is in no way involved, such provide little consolation for the practitioner when he is confronted with post-vaccination edema disease.

Most of the basic literature on this subject is found in European journals. Timoney[2,3] who has done considerable investigational work on the etiology of this swine disease, has concluded that it is noncontagious and appears to be of the nature of a toxemia. Luke and Gordon[1] reported their failure to isolate incriminating organisms from affected specimens and their inability to find toxins in the alimentary canals of pigs that had died. In a letter to VETERINARY MEDICINE, Timoney[4] stated that, "it (edema disease) is realtively easy to reproduce experimentally with the supernatant fluid of centrifuged intestinal contents given slowly through a very small bore needle into one of the ear veins." Timoney[5] also wrote, "that so far I have not succeeded in reproducing edema disease through the use of intestinal fluid from pigs other than those that had died of edema disease."

Since the etiology of the European outbreaks has not been established it should not be concluded that in this country we are dealing with a disease process of identical origin, although it does seem probable. Neither should we conclude that the same factor or factors are operative in all outbreaks in the United States. North American veterinary journals provide little to indicate that any major effort is being made in the Western Hemisphere toward determining the etiology of edema disease.

Case Report

Prior to the case herein described, the writer had diagnosed this condition on two occasions. One outbreak occurred about two weeks, and the other about two months, following vaccination. In both instances, losses were small and new cases did not develop following purging with epsom salts. In an outbreak seen recently involving a drove of approximately 150 pigs, symptoms and lesions were noted only in two pigs. Measures were not taken to prevent further occurrence.

On the morning of March 3, 1953, a drove of 93 pigs was vaccinated with a modified virus vaccine and hog cholera antiserum. Eight days prior to the vaccination, a new tenant had moved to the farm and had brought along 47 Hampshire pigs which had been farrowed during the last week in December. These pigs, in excellent health and averaging 50-60 lb., were added to an equal number of white pigs owned by the landlord. The latter pigs, farrowed during the first week in January, were of mixed breeding, apparently healthy, but many did not weigh over 25 lb. One pig of the landlord group died on March 1, the cause of death was not determined. Feed consisted of a blended mixture of commercial protein supplement, ground corn, and ground alfalfa supplied in a metal self-feeder. Pigs were confined on the concerte floor of a barn, the south end of which was open. Sleeping quarters were well bedded and dry.

Five pigs, all of the landlord group, were found dead 20 hours after vaccination. Three pigs of the same group exhibited marked incoordination and swelling of the eyelids. Temperatures of these were noted to be within the normal range. Autopsies were performed on four dead pigs. All revealed typical lesions of edema disease including deeply reddened areas over the inner surfaces of the upper forelegs, ventral thorax and abdomen, subserosal edema between the colon coils, marked edema in the stomach walls, hydroperitoneum, and enlarged, edematous-congested lymph nodes. The stomachs were filled with ingesta, the small intestines were comparatively empty, with congested mucosae, while the large bowels were filled with rather dry feces.

Following the diagnosis of edema disease, the feed hopper was closed for three hours

and the pigs were given a slop feed containing buttermilk, ground oats, and 5 lb. of epsom salts. After consumption of this material, access to the usual ration was again permitted. On the morning of March 5, three pigs were dead and two sick. One of the sick was killed and typical lesions of edema disease were noted. Three more were found dead on March 6 and the remainder were treated with a buttermilk slop containing 7 lb. of epsom salts. On March 7, seven pigs were found dead. By this time the owners were greatly alarmed and insisted that other measures be taken to stop the losses. As the trouble was confined entirely to the landlord group it was decided to separate the two groups and treat the landlord group with detoxifying tablets as had been done by Woods and Beamer[8]. On March 8, another pig was found dead and the remaining landlord pigs were treated orally with detoxifying tablets. One tablet per pig was added to the day's supply of drinking water, and it was suggested that the drinking water medication be continued for several days. It was clear at this time that most of the remaining landlord group, as evidenced by swollen eyelids, were suffering from edema disease. Following the loss of one more pig, during the night of March 8, losses from edema disease ceased in the landlord's group.

On March 10, one pig of the tenant group was found dead and several presented symptoms. The appearance of the sick pigs was striking in that there was massive edema of the ventral abdomen and jowls. Edema of the throat region was so extensive in three animals that respiration was seriously embarrassed. In addition, some pigs presented severe conjunctivitis, suggestive of hog cholera, and two pigs were scouring. Temperatures ranged between 103 and 106 F. The dead pig and one with severe dyspnea were autopsied. In addition to typical lesions of edema disease of massive character, the stomachs were found to be empty and the small intestines filled with watery, yellow fluid. By this time the landlord pigs apparently had recovered but were showing evidence of vaccination reaction.

It was apparent at this point that hog cholera as well as edema disease was coexistant in the drove. On March 11, one additional pig was found dead and the entire drove was started on a three-day sodium sulfathiazole threatment in a buttermilk slop at the rate of one grain per lb. of body weight. Detoxifying tablets also were added to the drinking water of the tenant group. On this date, three pigs of the tenant group, two live and one dead, and one live and one dead of the landlord group, were submitted to the State College Diagnostic Laboratory. Lesions of edema disease were found in all five pigs, and lesions of hog cholera were present in the living pigs submitted and in the dead pig of the tenant group. Later it was reported by the laboratory that attempts to isolate hog cholera virus from the specimens had resulted in failure. Two additional pigs of the tenant group died on March 11.

Although all of the pigs were markedly improved by March 12, with scarely any evidence of either edema disease or hog cholera, every pig was treated with 40 cc. of hog cholera antiserum. No deaths occurred during the following two weeks.

Etiological Investigations and Disease Reproduction Attempts

The last two pigs to die were autopsied and cultured by the writer. The medium used consisted of brain-heart infusion broth* containing sodium thioglycolate. The abdominal cavities were carefully opened, using aseptic technic and sterile instruments, portions of the livers, spleens, kidneys, edematous gastric tissues and mesenteric lymph nodes were removed and added to tubes of broth. Heart blood was removed with sterile syringes and added to broth medium.

After 48 hours at 37 C. cultures were examined. The heart blood, kidney, and some lymph node and edematous tissue cultures were negative for growth. Spleen, liver, and lymph node cultures apparently were contaminated with spore-forming rods and diphtheroids. Two lymph node and two edematous tissue cultures from one pig contained pure growths of staphylococci of the same type.

To examine the possible relationship of the isolated staphylococci to the edema disease process, a 20 lb., six-weeks-old Tamworth pig was obtained and given, orally, 10 cc. of a four-day-old broth culture of the organisms. Within 15 minutes the pig exhibited unmistakable signs of distress. The pig became restless, lying down and getting up, respirations became rapid and shallow and bristles on the back and neck stood up. Symptoms lasted about one hour and terminated in complete recovery. In six days, the experiment was repeated. This time symptoms were not elicited.

As it was felt that hypersensitivity might

*Difco Laboratories, Detroit, Mich.

possibly be a factor in the production of edema disease, the above pig was given three intraperitoneal injections of swine erysipelas antiserum in 5 cc. quantities at weekly intervals. Attempts to produce shock or symptoms of edema disease in two weeks following the last sensitizing serum injection resulted in failure.

Discussion

In speculating upon the unusual outbreak of edema disease described, one notes that the course of the malady in the two groups of pigs which were running together, is highly suggestive of an infectious and possibly a contagious process. A preformed toxin in the feed cannot be ruled out at this time, but it is difficult to accept such a hypothesis unless one is willing to accept also the probability of a state and period of sensitization followed by an explosive appearance of the disease. It should be noted that all of the pigs in the described drove were eating exactly the same feed and living under identical conditions for nine days before the disease appeared in the landlord's group, and that six days elapsed from that time until the syndrome was noted in the tenant's group. If a preformed toxin was involved, unless a sensitizing process was required, it is difficult to understand why the pigs of both groups did not react in a similar manner at the same time, or why the pigs of the tenant's group did not show symptoms sooner than the other pigs, since they obviously, on account of their size, were eating more feed. Regardless of what line of reasoning one assumes in trying to arrive at an explanation, obstacles are presented.

Before the new tenant moved to the farm the pig herd consisted of 94 pigs. Half of these were taken by the former tenant when he left the farm only two days before the arrival of the new tenant. The condition of the pigs taken by the former tenant was investigated about three weeks following the appearance of edema disease in the group described. The former tenant denied that he had lost any pigs.

Since, on numerous occasions, Timoney has been able to reproduce edema disease by injecting normal pigs with fluids from the bowel contents of pigs showing lesions of edema disease, but not with similar material from the intestinal contents of healthy pigs, it logically follows that in outbreaks in Ireland toxins or agents capable of producing symptoms of edema disease are present in the bowel contents of diseased pigs, and that sensitization to the agents is not required for the production of edema disease.

It seems likely to the writer that microbiological agents in the digestive tracts of diseased pigs are primarily responsible for the lesions and symptoms of edema disease, and with this in mind, it is deemed likely that a profitable line of approach might lie in investigations directed toward determining the microbiological floras of the digestive tracts of normal and of diseased pigs. Should it be found that pigs with edema disease consistantly harbor organisms which are not found or are found only in small numbers in normal pigs, a study of such organisms might profitably lead to solution as concerns etiology.

Summary

An outbreak of edema disease in a drove of 93 pigs is described. The drove consisted of two groups of unrelated pigs from different farms which had been added together only nine days before the disease outbreak. All, or essentially all, of the pigs of one group were involved with the disease process before symptoms appeared in the other group. Attempts at isolating an infectious agent and in reproducing the disease resulted in failure.

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Most important of proposed changes in the USDA budget is the elimination of federal tuberculosis and brucellosis indemnities effective as of July 1, 1954. This reduces budget requirements by nearly $700,000. but does not mean that there is any intention of discontinuing federal participation with states in the eradication programs designed for these diseases.

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A disease of nutrition, pregnancy disease of ewes, takes the greatest death toll of all diseases of sheep —*Frank Thorp, D.V.M.*

Anthrax In Florida†

JAMES E. SCATTERDAY*, D.V.M., M.P.H., ALBERT W. STICHKA**, and MILDRED M. GALTON§, Sc.M., Jacksonville, Florida

ANTHRAX in livestock in the United States, until recently, has been generally sporadic, with the exception of a few epizootic areas. Such areas are mainly in the lower Mississippi valley and in well-defined areas in southern California, Texas, Missouri, Nebraska, and South Dakota. Florida must now be added to this list.

The first recorded outbreak of anthrax in Florida occurred in a dairy herd near Miami in 1927 and 1928. The disease did not reach epizootic intensity and promptly subsided.

In 1935, near Quincy, Florida, anthrax was diagnosed as the cause of death in a small group of cattle imported from Kansas City, Missouri. Here too, the disease subsided without presenting any severe threat to the cattle industry. It is interesting to note, however, that a human death was reported as a result of this outbreak. The victim was a colored farm hand who contracted the disease while skinning an infected carcass.

During the years between 1935 and 1951 no cases of anthrax were reported in Florida.

Broward County, where the 1951 outbreak centered, lies on the east coast of Florida just north of Miami. Its western extremity extends to include a large expanse of range land that fringes the Everglades. This is an extensive dairy and beef producing area with herds as large as 4,000 cattle. These animals, as a rule, are in relatively poor nutritional condition in certain seasons and are plagued with anaplasmosis, acute parasitism, and mineral deficiency. Blackleg and malignant edema are not uncommon in this area. Since cattle losses among these herds are relatively commonplace, it is understandable that anthrax may have existed in Broward County for considerable time before diagnosis was ascertained.

The first clinical diagnosis of anthrax in the 1951 outbreak was made by a local physician. The patient was a cowboy who had suffered a minor knife wound while skinning the carcass of an animal which had died of an unknown cause. At the same time that the human case was diagnosed, a specimen of cow spleen was submitted to the Bureau of Animal Industry and to the Florida State Board of Health. From this specimen *Bacillus anthracis* was isolated. Promptly, the State Livestock Sanitary Board (now the Florida Livestock Board) dispatched a team of veterinarians to the area to determine the severity of the outbreak and to establish control measures to prevent further dissemination of the infection. A rigid quarantine on the movement of cattle was established, covering an area approximately 125 sq. mi., and with the aid of local veterinarians, all cattle and horses in this area were vaccinated with anthrax bacterin. It is estimated that 50,000 animals were given this prophylactic treatment.

Since the initial outbreak in October 1951, infection has been identified on 18 ranches and seven dairies in five counties with a loss of approximately 429 cattle. These infected herds represented more than 15,000 cattle. There have been a limited number of cases in horses and deer. One of the outbreaks on a large ranch was considered to be vaccination anthrax; five cows died and 18 recovered after treatment with penicillin. Predominantly, the disease has appeared in three adjoining southeast coast counties, Broward, Dade, and Palm Beach, with the exception of sporadic outbreaks in single herds near Jacksonville, Lakeland, and Gainesville, in Duval, Polk, and Alachua Counties, respectively.

An additional four human cases developed during the course of the outbreak. Of these, two were veterinarians actively engaged in postmortem examinations, and one was a female technician who handled specimens for culture, at the Broward County Hospital. The fifth case was a five-year-old boy whose mother was the nurse who had attended the cow hand and one of the veterinarians during their illness. It is presumed that the child was in-

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fected from contaminated clothing of the mother. All human cases responded to antibiotic therapy and made complete recoveries. Unfortunately the state laboratory was not able to procure anthrax cultures from any of the human infections.

Prompt control measures by members of the Florida Livestock Board and the cooperation of the owners have brought the number of cases occurring at present to a minimum. No new herds became infected in the epidemic area during 1953.

The history of one large beef herd is of interest. In October 1951, 150 animals of this herd of 450 died. Bacterin was used four times, but losses continued high. In March 1952, all animals were vaccinated with spore vaccine no. 2. The rate of mortality decreased slightly. One month later all cattle were again vaccinated with spore vaccine no. 2, increasing the dosage by 50%. This appeared to have no value. In May, spore vaccine no. 3 was used, resulting in an immediate decline in death rate. No new cases occurred until December. The number of cattle was increased to 1,500 in March 1953. At this time all animals were vaccinated with spore no. 3. The last case occurred in February 1953. Until June of that year, all animals sold went direct to slaughter. They are now allowed to be sold through the auction ring. All herd additions are vaccinated with spore no. 3.

At present, control measures enforced when outbreaks occur consist first of quarantining the premises for 60 days following the last death from anthrax. No movement of livestock from the infected pasture or pastures is allowed at that time. If there are few deaths and only one pasture, or adjoining pastures, is affected, animals for other pastures may go for immediate slaughter. However, if several pastures, which do not adjoin, are involved, the entire ranch is quarantined for the full 60 days. Spore vaccine no. 3 is used on cattle in infected pastures and usually on those in the adjoining pastures. It is not used on the entire ranch unless warranted by the methods used in handling the cattle, such as feeding, watering, or pasture rotation. No vaccinated animal may be moved for at least 21 days after vaccination. Just before release the animals are examined by the veterinarian in charge of anthrax control, Florida Livestock Board, and if any swelling persists, they are held longer.

In light outbreaks in dairy herds, bacterin and penicillin are recommended. The temperatures of all cows also must be checked prior to treatment. The initial dose of bacterin is followed by a second dose in two weeks, and a dose every 90 days thereafter. Infected dairies which have followed this procedure have had no further trouble. In one large dairy, anthrax occurred in a dry herd of 800 animals. Spore vaccine no. 3 was ineffective and it was necessary to use spore vaccine no. 4.

A supplementary regulation concerning the disposition of milk in anthrax-infected herds was passed by the State Board of Health in April 1952. The procedure is as follows:

"In herds where anthrax is known to exist, no milk shall be sold for seven days from the last known death from anthrax; or in lieu of this, the temperature of every cow shall be taken prior to each milking by a qualified, licensed veterinarian, approved by the local health department, and all animals showing any symptoms of illness shall be excluded from the milking herd; such animals to be held in isolation until released by the veterinarian; the milk from such animals being condemned during the isolation period."

Regulations required by the Florida Livestock Board concerning importation and sale of bone meal in Florida provide that:

"No bone meal, tankage, meat scrap, or any other animal by-products may be sold, offered for sale or used in stock feed or feed supplements in the State of Florida, unless such products have been so sterilized by heat as to destroy all anthrax bacilli and spores."

Early in the emergency a representative of the state laboratory was sent to Fort Lauderdale where, in the office of the County Health Department, facilities were established to screen specimens before shipment to the central laboratory. Fresh smears, obtained by making an incision in the animal and inserting a cotton swab into the splenic tissue, were Gram-stained and all specimens showing suspicious organisms were forwarded to the state laboratory at Jacksonville for culture and animal inoculation.

It was found that the most satisfactory technic for submitting specimens were merely to obtain a swab from splenic tissue and place it in the sterile screw-capped bottle, with convenient mailing container; supplied by all county health departments for use in the shipment of fecal specimens. The importance of these unbreakable, leak-proof containers is obvious. The splenic swab is far superior to a tissue sample as it allows the material to dry, thus enhancing sporulation and reducing the growth of putrefactive organisms that invariably swarm in specimens taken under field

conditions. When the material is contaminated with rapidly growing organisms of the spreader type or with organisms that are especially antagonistic to the growth or pathogenicity of the anthrax bacillus, laboratory diagnosis becomes quite difficult. A number of investigators have shown that organisms such as Pseudomonas, *Salmonella typhosa, Escherichia coli,* Friedlander's bacillus, and various staphylococci may be antagonistic to the anthrax bacillus, retarding its growth and inhibiting or destroying its virulence.

The diagnositic procedures used in the Florida State Laboratory follow closely the methods recommended by Dr. C. D. Stein of the Bureau of Animal Industry.

Upon receipt of suspected material a Gram-stain is made for microscopic examination. This procedure is of limited value as a large percentage of specimens received are contaminated with Gram-postive organisms which can not be distinguished reliably from *B. anthracis* by morphological characteristics. The anthrax bacillus shows considerable morphological variation, dependent upon the age of the specimen and upon its source.

A saline suspension is made of the specimen and approximately 0.3 ml. is injected subcutaneously into two mice. Simultaneously a loopful of the suspension is streaked onto several beef-infusion agar plates which are incubated at 37 C. overnight or for about 18 hours.

When suspicious colonies are located, they are picked and stabbed into semisolid agar by inserting the needle to a depth of approximately 1 inch. *B. anthracis* produces growth that is quite unusual and typical. After 18 hours incubation growth gives the appearance of an inverted pine tree. This is an invaluable aid in identification.

Mice injected with material containing anthrax organisms usually died in 24 to 48 hours. The incubation period may be longer, however. It has been the practice of the authors to give a negative report on the fifth day if the mice are alive and the anthrax organism has not been isolated culturally. The negative mice are held an additional five days before destroying.

On postmortem examination, there is relatively little pathology with the exception of a marked gelatinous edema on the abdominal wall. The spleen and heart are removed and direct smears are made of these organs. Again, a saline suspension is made and streaked onto beef infusion agar plates. When stained by Gram method, these slides usually show the spleen and heart blood to be teeming with anthrax organisms.

On several occasions, specimens from animals dead of *Clostridium chauvei* or *C. septicum* have been received. When mice are inoculated with pathogenic Clostridium they frequently die in from 24 to 48 hours. Stained smears from these mice may show Gram-positive rods in large numbers.

The recovery of Gram-positive rods, morphologically typical of anthrax, which reveal characteristic colonial formation on agar plates inoculated from the mouse viscera serves to confirm a positive diagnosis of anthrax.

The identification of *B. anthracis* in the State Laboratory may be condensed to four basic steps.

1. The finding of typical or strongly suspicious colonies on beef infusion agar plates.
2. The unusual inverted pine tree type of growth in semisolid media.
3. Positive virulence tests in mice.
4. The recovery of the organism from mouse viscera.

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Sex Hormones in Veterinary Practice

(Continued from page 184)

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All viruses follow the same laws of immunity and what can be done with one can be done with any other.—*Victor J. Cabasso, Sc. D.*

Advances In Animal Nutrition†

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In retrospect, advances during recent years in the science of animal nutrition have been phenomenal. These have made possible economical production of meat and profit to livestock growers.

These advances emphasize again the truths in the theory that the potential for growth and gains bred into our domestic livestock can be realized by improved rations.

THE use of antibiotics in the diets of animals is perhaps the most significant advance in the science of animal nutrition of this century. Apart from improved growth efficiency and economic returns, its use has opened entirely new vistas of research and understanding. It has brought the physiologist, biochemist, pathologist, veterinarian, and nutritionist into a common conclave of cooperation and mutual interest.

No longer can the several fields be divided into distinct circumscribed and restricted areas, where others "must fear to tread"; for now it is realized that all these others are, in fact, one. To serve the farmer, research in both nutrition and disease must operate in strict coalition.

There is no sharp line of division between the sick animal and the healthy; only a progressive and graded slope from the apparently healthy to the morbid or dying animal. There is not, in short, such a thing, under practical farm conditions, as the completely healthy animal. All animals are sick or diseased to some extent all the time; some obviously in poorer health than others, but all are in one way or another subjects of the ills to which flesh is heir. This, of course, is true of man also, but that is a problem for the medical profession.

Mode of Action of Antibiotics in Animal Nutrition

The mode of action of antibiotics in animal growth still remains to be completely answered, but our concept is much clearer today than even a year ago. There have been five suggested explanations as to how antibiotics function in animal nutrition. These are: (1) They function as nutrients—like vitamins; (2) they reduce infective agents, which, though not toxic, nevertheless compete with the host for essential nutrient components of the diet; (3) they stimulate growth of intestinal flora (microbes) which produce and synthesize essential nutrients such as riboflavin, vitamin K, niacin, *et cetera*, and thereby improve the nutritional level; (4) they enhance absorption of nutrients from the diet; (5) they destroy infective agents which poison or cause disease in the host.

Actually, with exception of the first point, all the remaining explanations are correct to some extent depending upon conditions and the biological environment of the host.

The early thought that antibiotics, fed in minute amounts, might act like vitamins was soon abandoned when it became clear that under conditions of strict sanitation, even approaching sterility, no antibiotic effect was observed. This has been repeatedly confirmed. The degree of sanitation can be estimated by the reduction in the antibiotic effect.

That antibiotics, even at low so-called nutritional levels, reduce intestinal microflora which compete with the host for nutrients in the diet, is well indicated by the sparing action of the antibiotic on proteins and vitamins. This value has been demonstrated repeatedly. It becomes particularly evident when nutritive components of the ration approach marginal levels, which explains why antibiotics show their greatest effect when the nutritional level of the diet is low; that is, the relative difference between plus and minus antibiotic is greater when the diet is submarginal, although the highest absolute yield is obtained, under farm conditions, when the antibiotic is applied to a diet of high nutritional level.

That antibiotics stimulate intestinal microflora favorable to nutrient synthesis is probable but difficult to prove, as the converse, cited above, could apply. However, it has been shown that antibiotics stimulate growth of certain intestinal flora known to produce nutritional end products. It also has been demonstrated that feeding these same bacteria to the

†Presented at the 36th annual meeting, Southern Veterinary Medical Association, Atlanta, Ga., Nov. 9-11, 1953.

host produces growth stimulation comparable to the low level antibiotic effect under equivalent conditions.

That antibiotics stimulate nutrient absorption has been demonstrated. Actual gut studies *in vivo* have shown enhanced protein, calcium, phosphorus, and manganese absorption. Recent trials with young swine have indicated that the addition of aureomycin® (chlortetracycline, Lederle) in the diet reduces the size of small intestine compared to nonantibiotic-fed controls. Thus, 46 gm. of antibiotic per ton reduced the small intestine weight 7.3% below controls, while 182 gm. per ton reduced it 25% below controls. Weight, average daily gain, and feed efficiency also was significantly enhanced over litter-mate controls. This is substantial evidence of increased absorption efficiency arising from antibiotic feeding.

As all evidence of antibiotic growth effect pointed to control of subclinical disease as the chief mechanism, it suggested that perhaps higher levels of antibiotic, particularly those of the wide-spectrum type, would give even greater growth efficiency effects. During the past year, a number of large-scale field trials have been carried out, particularly with poultry. Over-all conclusion has been inescapable —the major activity of antibiotics in animal nutrition is control of endemic disease.

Our whole concept of animal nutrition must be revised to appreciate that disease of variable quality and quantity is always present, and performs a continuous drain and stress upon the growth efficiency of farm animals. In many instances, it is not recognized by the grower until it has advanced to a degree where morbidity and even mortality is apparent. Then, of course, the veterinarian is called in and diagnosis and therapy instituted to reverse the disease and salvage the flock or herd.

Unfortunately, by this time the great bulk of the damage is already accomplished and many animals saved by therapy might better, from the economic standpoint, have been allowed to die. They invariably drag their feet, efficiency-wise, thereafter. This is particularly true of short-life meat animals, such as broilers and hogs.

It is clear now that prevention of incipient disease is the future goal of all sound animal production practice and not control of the developed syndrome. The therapeutic approach is only one of salvage, not efficient production.

One could consider our farm animals growing in a veritable sea of infective microbes, some lethal, others virtually innocuous, some frequently present, others rarely so, some in vast concentrations, others in low amounts; some highly specific as to method of infection, host relations, and etiology, others of a general nonspecific and variable nature; some distinctly sensitive to certain drugs, others comparatively resistant to all drugs. Between these extremes a continual shift in emphasis of infection occurs at different times on different farms with various animal species. It is this continuous variation, of struggle between the host animal and its parasites, which gives the vari-

Courtesy Dr. C. J. Whitehair

Fig. 1. This group of pigs is one lot of an unthrifty group used in a feeding experiment conducted in Oklahoma. These pigs received a basal ration of good quality. All had a chronic type of diarrhea. Appetites were poor.

able response to antibiotics under field conditions. It is this variation which is responsible for the widely divergent findings of experimental workers in the field as to which particular antibiotic is preferable for poultry, swine, or cattle.

As the feed manufacturer must attempt to produce a feed which is optimal under all conditions and not necessarily tailored to any given set of specific conditions, he has a major problem of selection. Actually, from the practical standpoint, his choice is the widest spectrum antibiotic available. That is, an antibiotic which is known to be effective in reducing growth and activity of the greatest number of microbial species, particularly those with clinical propensities.

The problem of the level of use is much more specific and one which must be attuned to the needs of the animal species and perhaps to the particular region and the particular major disease problems involved.

Thus in the case of chronic respiratory disease (CRD) of poultry, where the disease is endemic, a level of 50 gm. of wide-spectrum antibiotic per ton of feed on a continuous basis is indicated. Under such conditions disease may still "break" out under stress conditions and require higher doses temporarily to reduce its ravages within tolerance levels.

This problem of nonspecific and multiple infection in growing animals and its relation to the economy of growth efficiency is a new and highly fertile field of animal husbandry research. Already many significant advances have been made and a brief review of some of these salient and specific findings might prove valuable.

Antibiotics in Poultry Production

Since the first report of Stokstad and Jukes in 1949 relative to the growth-promoting effect of 5 to 10 gm. of aureomycin per ton of feed on growing chickens, an enormous amount of research on the antibiotic effect on poultry has been reported in techincal literature. Results have been varied, although it has been shown that at these low levels, penicillin, bacitracin, terramycin®[2] and aureomycin are all about equal in their over-all value. In some particular cases, penicillin appeared to be the most economical and effective, in others, bacitracin had the edge, again in other trials terramycn gave superior results and finally, aureomycin gave highest yields and superior conversions in other trials. Confusion and much argument reigned as each group of research men stoutly adhered to the particular antibiotic which had flourished in its particular trials. Feed manufacturers likewise had to make decisions; some did and selected a particular antibiotic which suited their conditions or economy or hunch. Others, equally confused, compromised by splitting the difference and using a little of each of several antibiotics in the hope of at least hitting on one which was right, and incidentally, hoping to satisfy all the customers. Now, after further research, we know that like the three blind men of Hindustan who went to "see" the elephant, all of them were right and yet all of them also were wrong.

The fact is that if subclinical disease is of low level and virulence probably any one or all of the antibiotics would give some growth promotion; but if disease level is high or of in-

®[2]Chas. Pfizer & Co., Brooklyn, N. Y.

Courtesy Dr. C. J. Whitehair

Fig. 2. This lot of pigs selected at random from the same group as those shown in fig. 1, were typical of those fed antibiotics with the basal ration. Note healthy haircoat and skin. Pigs in figs. 1 and 2 are the same age.

tense virulence, low levels or the narrow-spectrum antibiotics are largely ineffectual, while high levels of wide-spectrum antibiotics give sound economic return.

Today, in the major poultry producing areas, unfortunately, by and large the disease level is high, often very high. Air-sac disease (CRD), blue comb, nonspecific enteritis, and probably several other conditions, as yet not even crudely defined, are jeopardizing gratifying economic returns of the industry. Against these massive infections lying between subclinical and clinical intensity, low feeding levels, particularly of narrow-spectrum antibiotics, are virtually useless.

A large number of field trials employing high levels of wide-spectrum antibiotic in the feed, on both a continuous and intermittent basis during the past year, in the concentrated poultry areas of Delaware and Georgia, have demonstrated economic value.

Table 1 shows the effect of high-level antibiotic on the growth of broilers at four weeks of age, two weeks after chronic respiratory disease had been introduced into half the pens. Clearly, although no apparent disease symptoms had developed, the infective agent was holding back the non-antibiotic-fed birds significantly, while on the high level of antibiotic, growth was as good among the infected birds as the uninfected control.

TABLE 1. Showing Effect of Applied CRD on Weight of New Hampshire Broilers 14 days after Inoculation in Relation to Aureomycin in the Feed. (Data lb./M birds)

CRD Applied	Aureomycin Nil	fed 50	grams/ton 100	CRD Av.
No	720	768	745	744
Yes	623	714	767	702
Aureo av.	671	742	756	723
Least difference @ 5% pt.				
Aureomycin means	42			
CRD means	34			
Aureo. X CRD	60			

Table 2 gives the same flock of birds, the data being expressed as feed conversions, *i.e.*, lb. feed/lb. gain. Here again is demonstrated that the disease depressed growth efficiency in the absence of antibiotic but failed to do so in its presence.

This experiment, conducted on 7,200 birds, was repeated on another flock with essentially the same results. The conclusion was inescapable; the disease was debilitating the flock inconspicuously and subclinically, but in a manner which would be only too apparent when the flock was marketed and the returns computed. The antibiotic reversed this trend and sustained the growth efficiency in the face of massive subclinical infection.

TABLE 2. Feed Conversions of New Hampshire Broilers Grown on Variable Levels of Aureomycin as Affected by CRD (Data lb. feed/lb. gain)

CRD Applied	Aureomycin Nil	fed 50	grams/ton 100	CRD Av.
No	2.08	1.94	1.98	2.00
Yes	2.34	2.10	2.03	2.16
Aureo. av.	2.21	2.02	2.01	2.08
Least difference @ 5% pt.				
Aureomycin means	0.22			
CRD means	0.18			
Aureo. X CRD	0.31			

In these experiments, birds and feed consumed are weighed every two weeks. This allows for determination of weight gains and feed conversions, period by period, of two week's duration. Analyzing the data expressed in this manner, reveals that the high level antibiotic consistency accelerates growth despite disease right up to the terminal period before market, when the nonantibiotic-fed birds really suffer and exhibit a significantly depressed feed conversion and coincident drop in weight gain.

An interesting field trial was put into operation when a large Delmarva grower suffered a severe outbreak of CRD in a flock of 20,000 Barred Rock crosses at six weeks of age. The flock was divided into five parts and treated in the five ways indicated in table 3. Resulting yields and conversions are significant in that they show that the antibiotic substantially reduced the deleterious course of the disease, even though it was well started before the antibiotic was added to the ration. However, much higher levels were needed to break the disease attack than would have been needed to prevent it.

The important point in these data is that although improved weight gains and reduced mortality were substantial, the savings in feed conversion were phenomenal and clearly reveal the site of real damage from disease on growth efficiency. It also should be recorded that the regular (control) feed this grower was using contained the so-called "nutritional" level of antibiotic. It was obviously ineffectual in this case of a rampant endemic disease.

TABLE 3. Effect of Various High Levels of Aureomycin on a Natural Outbreak of CRD in Broilers

Treatment Aureomycin fed gm./T.	Mkt. Wt. lb.	Mort. %	Feed Conv.
Control	3.29	10.9	4.75
400 gm. 14 days			
100 gm. to market	3.59	6.1	2.90
200 gm. to market	3.46	5.1	3.57
100 gm. to market	3.27	5.7	3.69
Soluble aureomycin 250 mg./gal for 14 days only	3.43	7.8	3.86
Least differences for weights @ 5% pt. 0.09 @ 1% pt. 0.12			

Another similar situation prevailed in North Georgia on a large commercial broiler farm where some ten houses each of 10,000-bird capacity are continuously rotated about one week apart. Continuous respiratory infection (identified as CRD) had plagued the entire farm for over two years when a high-level antibiotic trial was installed.

Table 4 shows the pertinent data from this trial. The high-level antibiotic significantly improved growth, livability and conversion and paid for itself by over $100./M birds at 1953 prices.

Incidentally, both farms, the one in Delaware (outlined in table 3), and the one in North Georgia (table 4), now routinely use 50 to 100 gm./ton of wide-spectrum antibiotic in the feed continuously and results have been most satisfactory from the profit standpoint.

TABLE 4. Results Secured from Continuous Feeding of 100 gm. of Aureomycin to Broilers Grown in N. Georgia Under Conditions of Endemic CRD

Item	Control	100 gm. Aureo./Ton	% Gain
Average Wt. lb.	2.87	3.08	7.3
Total Wt. lb.	12,563	14,948	
Av. wt./bird set	2.51	2.90	13.5
Mortality	10.68	2.82	73.6
Culls	1.3	0.1	90.8
Feed Conversion	3.11	2.81	9.64
No. birds started	5,000	5,000	
Breed: Vantress crosses			
Age Marketed: 10 weeks			

These findings have been replicated on turkeys in relation to enteritis and blue comb, as well as sinusitis, and in repeated trials on chickens, including laying and breeding stock. In the latter, significant increases in both egg production, hatchability, and livability of progeny have been secured. Further work on mature birds is needed, but the trends to date are in the same general direction as with the young, growing birds. Improved growth rate, reduced mortality, improved feed efficiency and production have been secured repeatedly under both commercial, practical, as well as strict experimental conditions, on many hundreds of thousands of birds, through the judicious use of a high level of wide-spectrum antibiotic in the face of incipient disease.

It would seem, therefore, that, at least in areas where these diseases are endemic, high-level feeding of these antibiotics will become standard practice to assure economic returns.

Antibiotics in Swine Nutrition

This subject was reviewed completely to date (March, 1953) by Braude, Wallace, and Cunha of the Florida Experiment Station[1].

Five antibiotics have been studied and found to have some efficacy in promoting growth in swine. See following table:

	Growth Index*	Feed Conversion Index**
Aureomycin	135.9 (187)***	90.2 (146)
Penicillin	110.6 (53)	94.3 (44)
Streptomycin	115.2 (50)	94.4 (41)
Terramycin	123.7 (23)	93.7 (17)
Bacitracin	109.0 (12)	103.0 (10)

* Percentage over control.
** Percentage of control
*** No. of trials reported.

Here again, the wide-spectrum antibiotics in the over-all have induced higher growth and conversion gains over controls, although in certain areas and conditions some of the others have shown good growth-promoting effects. The narrow-spectrum antibiotics have shown growth promotion in hogs averaging 10% above controls; while the wide-spectrum types have promoted growth an average of 15% over controls or about 50% more than the narrow spectrum types.

Growth promotion from antibiotics is invariably greater on runt pigs and an average of 12 comparisons showed growth stimulation among runt pigs of over 82% versus controls.

It is noted that although improved early growth stimulation from antibiotics is marked, it is likewise significant at later ages, even during market fattening. In fact, the bulk of the evidence indicates it is unwise to remove the antibiotic at any time during either growing or fattening.

It is of interest that the antibiotic effect on swine is found among hogs on pasture in similar degree to those in dry lot.

Significant improvements in early growth, feed efficiency, and livability have been achieved by using wide-spectrum antibiotics in milk replacers and creep feeds for baby and suckling pigs. Indeed, the milk replacers fortified with antibiotics and vitamins, bid fair to replace completely the dam after one week of nursing, increasing growth and viability of the progeny and returning the dam to the breeding pen more rapidly and in better condition.

Knowledge of the value of antibiotics on pregnant sows is incomplete, but some recent data reveals increased weights of newborn pigs, numbers weaned, and weaning weights.

The optimal levels of aureomycin in feed for hogs is considered to be:

Pregnant and lactating sows	5 mg./lb.
Pig Starters (creep feeds)	25 mg./lb.
Growing feeds up to 75 lb.	10 mg./lb.
Growing feeds up to 75—200 lb.	5-10 mg./lb.
Concentrates—35% protein	25 mg./lb.
40% protein	35 mg./lb.

In all cases, the mode of action of antibiotics among swine is considered essentially related to improvement in intestinal flora with particular reduction in the number and virulence of enteritic agents causing scours.

Antibiotics for Ruminants

Research to date on the use of antibiotics for calves has been reviewed extensively by Knodt[2].

In general, it was found that only the wide-spectrum antibiotics were effective in decreasing incidence of scours, increasing feed efficiency and growth rate among dairy calves.

Here again, convenience and efficacy have been achieved by feeding the antibiotic in milk replacers. Knodt showed that 2 gm. of aureomycin per 100 lb. of milk replacer was the optimal level.

Rusoff showed that the antibiotic was more effective in maintaining growth rate and reducing scours when the starter feeds contained high quality soybean or cottonseed meal as protein source.

Matsushima has found that antibiotic tablets (containing 20 mg. of aureomycin) fed one per calf per day, significantly improved growth and feed efficiency and reduced scours and liver abscesses.

Jacobson, at Iowa, determined that body weight gains of calves fed aureomycin from birth to 200 days, continuously, were superior to those fed either from birth to 11 days or from 116 to 200 days which, in turn, were superior to control.

This indicated that the antibiotic at the levels fed (18 gm. per ton) in no way depressed the growth and development of favorable rumen flora.

Recent work at the Purdue Station and in our laboratory has shown that antibiotics may be of significant value in the fattening of beef steers and lambs. This is surprising as it had always been assumed antibiotics would interfere with adult rumen flora and depress daily gain. The Purdue workers, however, report that the feeding of 75 mg. of aureomycin per head per day actually improved daily gain and feed efficiency and reduced cost per pound of grain by 3¢ on a high roughage diet, where rumen activity would be greatest. We found precisely the same thing with fattening lambs.

Conclusion

Antibiotics are now a solid component of animal rations; they will remain an important adjunct of the feed industry.

Their action is clearly one of disease control, by reduction of incipient infection, be it of high (clinical) or low (subclinical) degree.

For this reason, the wider the spectrum of the antibiotic, the more likely it will be effective over a wide range of farm conditions and usage.

Such antibiotics have to be used at levels commensurate with the type and intensity of the infective agent they are intended to combat.

In poultry, high levels (50 to 100 gm./ton) have proved effective in sustaining growth efficiency and economic reutrns in the face of serious infections (air-sac disease—CRD, blue comb, enteritis) of both growing and laying chickens and turkeys.

In swine, wide-spectrum antibiotics have been shown to be economic and efficient for creep feeds and milk replacers in young pigs, and as additives in fattening rations and diets of pregnant or lactating sows.

In ruminants, the wide-spectrum antibiotic aureomycin has been shown to enhance growth efficiency and reduce scours in young calves, and also to promote improved growth and lower costs in fattening steers and lambs.

Antibiotics have opened an entirely new era in animal production.

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(*Continued on page 198*)

Some Equine Practice Problems†

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THE subject of equine practice still remains an important part of veterinary services even though the mechanical age has replaced the working animal of farm and commerce. The general practitioner today may find his equine patients few and far between unless his practice area includes race tracks, breeding farms, or training stables. Exhibition and light riding horses require the services of only a few veterinarians. In preparing this brief discussion I felt that since the demand for veterinary service to the horse requires such a small part of practice time of the general practitioner, I would confine the discussion to a few unusual conditions presented in practice.

Wounds of the Mouth

Wounds of the mouth, fortunately, are generally of minor importance and most heal rapidly under ordinary care. Those requiring special attention are ones involving bony structures, the most common being injury to the intermandibular space. This usually is caused by too severe curbing and rough handling by riders. Such wounds vary from bruising of the periosteum to fracture of the mandible. Ordinary injuries respond by removing the cause (*i.e.* the bit) while those complicated by fracture, removal of bone fragments or curettage must be undertaken. The average trainer feels that, after such treatment, he can replace the bit and put the horse back into training. Constant irritation on the traumatized area creates exuberant granulation that will not heal unless carefully removed surgically.

In our practice, removal of such granulations may be done with scapel or electric cautery. Daily application of copper sulfate (blue stone) however, does a better job in less time and has the advantage of owner participation in treatment. Copper sulfate acts only on excessive granulation and does not destroy new epithelial cells at the edges of wounds. By reducing the exuberance, these cells can cover the granulating surface and suppress further growth. Wounds on the tongue and cheek result, generally, from points on teeth. These are corrected by floating or cutting. Molar caps must be removed from young saddle horses.

Suturing accidental wounds has always presented a problem. The decision to suture or treat as an open wound depends on many new factors revealed by new findings in wound healing. Owners expect all wounds to be

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sutured regardless of the body part involved. With the exception of wounds below the knee and hock, attempts should be made to close gaping wounds. On leg wounds, our results are most satisfactory with compression bandages, supported by tape, for at least 24 hours. The problem of self molesting dressings is most important and the use of a cradle, or tie, that will prevent the animal from reaching the area is a necessary aspect of treatment.

Many good horses are injured in transportation between exhibition points, many resulting from slipping off or through the tail-gate, or ramp, while loading. The hock and fetlock of the hind legs are most often the site of such injuries. These wounds present the problem of excessive granulation. Protecting such wounds from self-injury and avoiding use of antiseptics that tend to stimulate granulation tissue is most important. Daily cleansing with mild boric acid or epsom salts solution, and the application of furacin ointment, under light dressing, is our usual procedure. When exuberant granulations develop, these are removed surgically or by cauterization.

Lameness

Emphasis of owners and trainers on high action and the means employed to develop it in both saddle and light harness horses, results in a common "man-made" lameness. It is not uncommon to find from five to ten pound weights placed on each forefoot of fine show animals; this in addition to long toe and low heel shoeing, which often so changes the normal axis of the lower phalanges that permanent repair is impossibe. Application of excessive weights is the principle, predisposing cause of at least 90% of lameness below the knee, when such practices are followed. The problem of correction is complicated by the reluctance of trainers to sacrifice aids for developing action. They expect all other means of correction to be exhausted first. In most instances, this is impossible—foot weights must be removed gradually and attempt made toward correction of angulation.

Mechanical Disturbances of Respiration

Under this heading I place most conditions not associated with systemic involvement, including sinus disorders, tumors of the nares, paralysis of the laryngeal saccule (roaring), and other miscellaneous anomalies. After diagnosis, corrective surgery is recommended.

At this point I would like to mention distress exhibited by saddle and harness horses on both inspiratory and expiratory phases of breathing while ridden or driven. When in motion it frequently is observed in horses with extremely well-set heads that the head takes a nose-in position, causing muscles around the throat and back of the tongue to push up into the pharynx. Most of these animals are noted to breath normally if examined in a trot on a longe line or lead.

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Continuous Phenothiazine-Salt Mixture for Control of Stomach Parasites of Sheep

A eight-year study[a] conducted under practical range conditions of the Edwards Plateau of Texas indicates that the large stomach worm, *Haemonchus contortus*, of sheep can be controlled satisfactorily through the continued use of 1:9 phenothiazine-salt mixture supplied free-choice as the method of treatment.

No evidence of toxicity was observed during these extended experiments. Many of the animals in the treated flock attained the age of six to eight years before marketed and never had received any salt except that contained in the mixture. Rate of consumption was noted to be slightly less than ⅛ lb. per month, varying from month to month and from year to year. Variations in amounts consumed were attributed to the presence, each year, of growing lambs in pastures and to annual rainfall of the region.

Comparison of worm-egg counts in the treated and untreated flocks, larval development in feces, and actual worm counts at autopsy indicates that the 1:9 phenothiazine-salt mixture satisfactorily controlled stomach worm infection in range sheep under management practices as applied in the region. These findings may be applicable to similar arid areas but may need to be modified under varying climatic conditions, particularly greater rainfall.

[a]Price, D. A., Hardy, W. T., Boughton, I. B., Phenothiazine-Salt Mixture, Free Choice, for the Control of the Large Stomach Worm in Range Sheep. Texas Agricultural Experiment Station bulletin 708. College Station, July 1952.

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(*Continued from page 196*)

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SMALL ANIMAL PRACTICE
V M

Parasitic Diseases Of Animals And Their Relation To Public Health†

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Authorities have emphasized repeatedly that helminth parasites cause unestimable damage to host animals and loss to growers by reason of the insidious action of lesser understood stages of the metamorphosis of these nematodes. Further, they maye be of considerable importance from a public health aspect. In this article, the author outlines what is known about visceral larva migrans, a newly recognized syndrome closely related to creeping eruption in man and due to migration of dog ascarid larvae in an unnatural host. This type of parasitism may be a common cause of certain diseases of obscure etiology in both man and animals. Whereas a few years ago worms of other animals were considered innocent in man until proved guilty, today it appears reasonable to regard them all as guilty until their innocence is firmly established.

PARASITISM may be viewed either as a natural or as pathological relationship. When viewed as the former, one thinks of the parasite as a species of organism which, through evolutionary changes, has adapted itself to a dependent life on, or within, one or more host species; the host species likewise having undergone evolutionary changes. Thus we may speak properly of natural, or normal parasites of individual host animals and normal hosts of the individual species of parasites. In general, parasites depend largely upon fortuitous passage from host to host. Therefore, in order to guarantee a succession of generations, they tend to produce enormous numbers of infective stages which are cast into the natural environment of the host. Under natural conditions only a small percentage of these cysts, eggs and larvae succeed in completing their life cycles. Due to various forms of accidents, most of them perish. In considering the parasitic diseases of animals in relation to public health, the most important type of accident that may befall the infective stages of parasites is to get into some unnatural host such as man.

In the United States, human parasitic infections that are commonly, or exclusively, acquired from other animals include such examples as trichinosis, echinococcosis (hydatid disease) taeniasis, (pork and beef tapeworm infections), dipylidiasis (dog tapeworm), hymenolepiasis (dwarf and rat tapeworm infections), cysticercosis, balantidiasis, and toxoplasmosis. There is not much to be added to the general textbook information on most of these infections. Trichinosis continues to be a public health problem, but it is hoped that the control of the feeding of garbage to hogs may prove effective in reducing the infection. There has been no noticeable recent trend in the incidence of beef tapeworm, but the pork tapeworm (*Taenia solium*) seems to have become rare in the United States. However, cysticercosis in hogs, *i.e.* measly pork, which presumably is contracted from *T. solium* in man, is still seen occasionally[13]. This interesting and important epidemiological paradox merits vigorous and intensive study whenever the opportunity is offered.

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Several recent reports have called attention to *Echinococcus granulosus* as a potential danger in this country. In Canada, Miller found evidence of recently acquired autochthonous hydatid infections in 141 persons and recovered the adult *E. granulosus* from 28% of 114 dogs[11]. About a year ago, in the vicinity of Clarksdale, Mississippi, Franklin and Ward found two of 50 dogs infected, and more recently discovered two more[6,7]. The discovery of four naturally infected dogs within a short period in Mississippi, whereas only three such infections in dogs had previously been reported from the entire country, should stimulate more intensive search for Echinococcus in dogs of other areas.

Of all the animals associated with man, the dog is probably the most important as a source of parasitic diseases. In some regions the most common infection derived from dogs is creeping eruption, or cutaneous larva migrans. The only widely recognized cause of creeping eruption is *Ancylostoma braziliense*, one of the several species of hookworms of dogs and cats. This hookworm is common in the coastal plains of the southeastern United States from Virginia to lower Florida. The principal reason for regarding *A. braziliense* as the chief etiological agent of creeping eruption is that the disease is most prevalent within the known geographic range of the worm. However, *A. braziliense* is not the only species involved in creeping eruption. *Uncinaria stenocephala*, also a dog hookworm, has long been known to be capable of penetrating human skin and producing characteristic serpiginous lesions, and more recent studies have shown that *A. caninum*, which is the most common of the hookworms of dogs and cats throughout the United States, as well as the cattle hookworm, *Bunostomum phlebotomum*, may produce this type of infection. Generalizing from these observations, creeping eruption may be viewed as a type of parasitic infection resulting from various species of nematode larvae which in their normal hosts reach the intestine by a migration route from soil to skin, through the skin to blood stream, to the lungs, trachea, pharynx, esophagus, and finally the intestine. When these larvae penetrate into the skin of man their further migration to the blood stream and lungs is delayed and they move about for a more or less lengthy period in the outer layers of the skin, setting up a reaction which marks the path of their seemingly aimless wandering.

A new clinical syndrome, closely related to creeping eruption and one which likewise involves dogs and cats now is in process of being defined and understood. Under the name visceral larva migrans it was first described in 1952 by workers in New Orleans, Louisiana[4]. In the short period since, it has been reported from many other localities. As a disease of unknown etiology, it was earlier reported under various designations such as familial eosinophilia, Weingarten's disease Frimodt-Möller's syndrome and eosinophilic pseudoleukemia. Including the more clearly recognizable cases among the latter and some unreported recent ones, the infection is known to occur in 12 states representing all sections of this country, in England, and in Puerto Rico. It is safe to predict that eventually it will be recognized in nearly all parts of the world, especially in areas where the climate is warm and children live more or less intimately with dogs. While there is much yet to be learned about visceral larva migrans, it is already clearly evident that veterinary clinicians, especially those who are in small animal practice, will be called upon to assume an in-

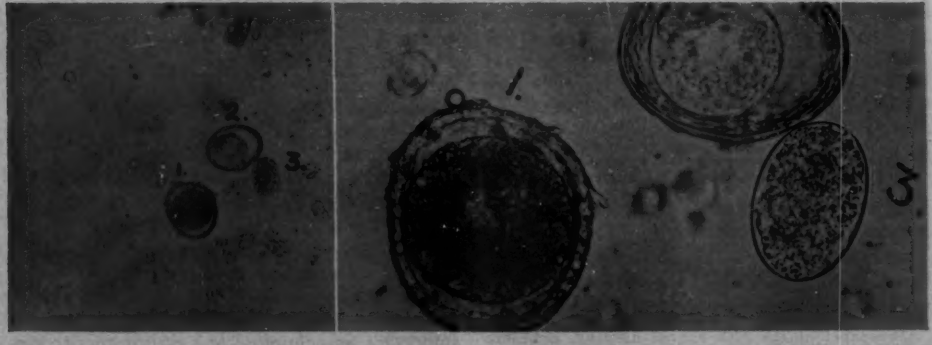

Fig. 1. Parasitic ova observed in a single microscopic field of a preparation made from the feces of a dog. (1) *Belascaris marginata*. (2) *Toxascaris limbata*. (3) *Uncinaria stenocephala*. (Left) Low power - x 100. (Right) High power - x 400. After Benbrook.

creasingly important role in the protection of the individual and the public health.

Like creeping eruption, visceral larva migrans is the invasion of human tissue by larvae of nematode species that normally reach maturity in non-human hosts. The two conditions differ in that in the one the skin is invaded and damaged while in the other the liver, lungs, kidneys, brain and other internal organs are affected. It already has been stated that hookworms are mostly responsible for creeping eruption. Visceral larva migrans apparently is most commonly caused by *Toxocara canis* and *T. cati*, the large ascarid roundworms of dogs and cats respectively. The life cycle of Toxocara species in the normal host is essentially the same as that of the pig ascarid, *A. lumbricoides*. It involves the production of an abundance of heavy-shelled eggs that reach the soil through fecal contamination, the development of infective-stage larvae within the egg, the ingestion of soil containing the infective eggs and the hatching of those eggs in the intestine. The larvae then migrate through the intestinal wall via the vascular system, to the liver, lungs, trachea, pharynx and back to the intestine where they mature and begin egg-laying in three or four weeks. Now if a home is visualized as housing a small child and a young dog, and it is taken into account that the child and the dog are by nature about equally unhygienic, it is not difficult to imagine various means of transfer of infective stages of the dog's parasites to the child and the child's parasites to the dog. This type of reciprocity is sometimes harmful to the dog (amebic dysentery in dogs is sometimes fatal) but it more frequently is harmful to the child. Of the several helminthic infections contracted from dogs, among the most serious known to date is visceral larva migrans caused by *T. canis*. Probably it is also the most common.

Knowledge of Toxocara in man is based on studies of numerous liver biopsy specimens and two postmortem examinations as well as published and unpublished observations on over 30 clinical cases. When Toxocara larvae are freed from the egg in the human intestine they migrate to the viscera, following the same route as in the dog. Instead of passing from the lungs to the intestine in a more advanced stage of development, as they do in dogs, if they return to the intestine at all they are still in the infective stage and merely repeat the visceral migration. The details of how the larvae distribute themselves at different periods of the infection are not yet known. Judging from observations on the fatal cases,[4,8] from studies on experimental animals and two human volunteers,[13] it appears that for the first few days most of the larvae are in the liver. Afterwards they tend to move to the lungs and from there to various other organs, especially the kidneys, brain, and spinal cord. Some of the larvae become encapsulated and destroyed within a month or so, but most of them live and continue active migration through the tissues for many months. There is good evidence that a high percentage of them remain active for considerably longer than a year. Absolute diagnosis of this infection can be made only by finding and identifying the larvae in tissues taken by biopsy or at autopsy. However, the disease sometimes can be recognized by characteristic clinical and laboratory findings[10].

The damage produced by Toxocara larvae in human tissues varies with the number of larvae involved, the duration of the infection and the particular tissues invaded. The disease then varies from subclinical to severe, and occasionally may lead to death. In one fatal case, death apparently was due to overwhelming invasion of many of the organs including the heart and brain by an inestimable number of larvae of *T. canis*. The more usual case is one in which the illness is mild, tends to be recurrent, and is always of long duration. The chief manifestations are fever, fretful disposition, whimsical appetite, and general poor health. Infiltration of the lungs usually is noted and a high persistent eosinophilia is always a conspicuous feature of the syndrome. Individual larvae may produce extraordinary damage if they invade particular tissues such as nerve centers of sense organs. In 1950, nematode larvae were discovered in a number of diseased eyes that had been removed from children because of suspected retinoblastoma[14]. It is not yet certain whether any or all of the worms found in the eye were Toxocara but they were indeed nematode larvae and the disease produced by them is in all known respects a form of visceral larva migrans. Ascarid larvae, probably *T. canis*, twice have been found in the human brain[1,6].

Visceral larva migrans occurs mostly in children under three years of age. Inasmuch as infection is acquired through eating soil that was earlier contaminated by dogs, children in the dirt-eating stage of their development would be expected to be most frequently and most heavily infected. For the reason that no effective treatment of visceral larva migrans has been found, it is especially important that preventive measures be used to the fullest pos-

sible extent. The following are suggested for consideration:

1. Children may be prevented from eating dirt. This of course is difficult to enforce and at best can be only partially accomplished.

2. Toxocara eggs in the soil may be destroyed by working the surface to hasten drying; or the top soil may be turned under to put the eggs out of reach of children. No chemical is known to be useful in killing the eggs in soil.

3. Dogs can be kept free of ascarid worms. While this recommendation is feasible, it requires constant and frequent attention. If the dog is allowed any out-of-doors freedom, stool examinations and/or treatment will be required every three to four weeks because infections are readily acquired and the worms usually mature in less than a month.

4. Families with small children can dispense with pets, especially young puppies and kittens which are more apt to be infected than are the older animals. However, children in families who have no pets at all are not safe in most neighborhoods. Milburn, in California, reported a heavy infection in a two-year-old child who had no known contact with dogs and cats[10].

5. Dogs and cats could be put under more rigid control. Citizens of well organized communities, by all standards of decency and neighborly conduct, should be restrained from deliberately encouraging their dogs to defecate in their neighbors' dooryards.

6. As a general recommendation, it would seem reasonable to suggest that, at least in urban communities, children and pets should be given equal care regarding periodic health examinations, immunization scheduling, cleanliness and hygienic training.

To meet his responsibilities fully in an effort to protect the public health, the veterinary clinician must have precise and complete knowledge of all the parasites of dogs and cats in his community. Not all of the dog and cat parasites are known to be harmful to children but few of them are known to be harmless. Considered from the standpoint of public health, the most important worm parasites of dogs are *Toxocara canis, Ancylostoma braziliense, A. caninum, Uncinaria stenocephala, Strongyloides stercoralis, Echinococcus granulosus, Dipylidium caninum* and possibly Dirofilaria spp. The normal life cycle of these parasites and their relation to human disease are too well known to require further review here. However, because there is new interest in *T. canis* as a cause of visceral larva migrans, certain facts regarding it may be listed. *T. canis* is found in dogs and other species of Canidae in all parts of the world. It is especially common in younger animals and frequently is observed as cream, or flesh-colored, round, unsegmented worms up to six inches in length, in their vomitus and stools. In some regions Toxocara is so common that dogs rarely escape infection. It matures in the small intestine after three to four weeks prepatency, and for a period of a few months it lays enormous numbers of large spherical eggs with heavy, bile stained, pitted shells. In warm, moist soil the eggs become infective in about two weeks (they are not infective in fresh feces); they may remain viable for a year or more but they are killed by direct sunlight, drying or freezing. Studies conducted in New Orleans and Georgia demonstrated that Toxocara eggs occur frequently and abundantly in dooryard soil[7,8,9]. Although the old dogs usually have few if any mature worms in the intestine, they regularly acquire new infections from soil and have larvae in their tissues. By this means they infect their young prenatally. Vermifuges are fairly effective in removing *T. canis* from the intestine but none of the drugs presently in use can be depended upon to eradicate an infection with one treatment. Therefore, post-treatment stool examination is a necessary routine.

Innes and Shoho have called attention to the possibile public health significance of another larva migrans-like disease[9]. It has been found in Japan and Ceylon that a filarial worm (*Setaria digitata*) which lives normally, and generally harmlessly, in cattle produces serious and sometimes fatal disease in sheep, goats, and horses. In the cow, the worm lives in the abdominal cavity, but in abnormal hosts it tends to invade the central nervous system and the eye. Setaria species occur in domestic animals in the United States as well as other parts of the world. Inasmuch as the infective stages are transferred by mosquitoes, human infection would seem to be a possibility. To date, however, no proof has been offered that it does or does not occur in man. Nevertheless the observation is of interest and significance, because it further points out that the immature stages of worm parasites may be far more pathogenic in abnormal hosts than they are in their normal hosts, and owing to the difficulties of diagnosis, this type of parasitism may be a common cause of certain

(*Continued on page 205*)

Management Of The Leptospirosis Patient†

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THE management of the leptospirosis patient is essentially no different than the management of any other acutely or chronically ill medical patient. Therefore, this paper may prove more than a little disappointing to those who hoped to read of a new and completely successful treatment, outlined in specific order of preparation and specific doses. Also, giving the excuse of the press of time and the economic demand for immediate results, one may look longer and more often at the drug shelf than at the patient. Nor do I apologize for the use of the word excuse, since I feel that mental laziness is more often than not the actual cause. Obviously, from the definition, no treatment can be justified that has not been preceeded by a thorough and intelligent appraisal of the nature and extent of the existing pathology. Whether the final diagnosis be pneumonia, enteritis, or leptospirosis, it must involve an appreciation of the total pathology of each organ and system within the body. In other words, before management, a diagnosis. Not just by name, but tissue by tissue.

What, then, can be said for the management of an animal allegedly infected with Leptospira. Let us consider first the acute syndrome, or so-called septicemic stage. Presented is an animal infected by a microscopic parasite which is circulating freely in the blood stream, and in most of the tissues. While doing so, it voids its own metabolic by-products and perhaps also may liberate a rather powerful exotoxin. It is with the effects of these toxins that we must cope in addition to attempting to destroy the invading organism itself. Leptospira seem quite vulnerable to both streptomycin and penicillin given in large doses and continued for four to six days after the animal exhibits clinical evidence of recovering. Other antibiotics have been tried in this condition. It has been found in golden hamsters that aureomycin[®1] was effective when commenced within 32 hours after experimental infection. Terramycin[®2] was effective when given up to 48 hours after infection, while chloromycetin[®3] had no therapeutic effect.

Toxemia

Relief of toxic symptoms, however, is considerably more complicated. Toxins may produce hemolysis of red cells, resulting in one form of jaundice. These toxins may alter capillary permeability, resulting in the petechial hemorrhages observed in the mucosa. If these effects are severe, then whole blood transfusions would be indicated, in amounts up to 10 cc. per lb. of body weight. Vomiting is quite common, especially in the later acute stages. When severe, it results in a considerable loss of chlorides. This occurs because chlorides are drawn from the extracellular body fluids to replace electrolytes lost. In such case administration of normal saline solution may be indicated in doses of 2 to 10 cc. per lb. of body weight by the intravenous, subcutaneous, or intraperitoneal routes. Part of the body defense mechanism has established an elevated temperature, which results in about a 7% increase in metabolic rate for each degree rise. This temperature rise serves to increase phagocytosis as well as antibody production. However, increased metabolic rate also has its side actions. For one thing, it greatly increases the utilization of the body stores of glycogen, depletion of which is fuel added to the fire. When fats are consumed without glycogen, ketosis and acidosis result. This is prevented quite easily by carbohydrate administration, either as karo syrup or glucose solution orally, or glucose 5% solution parenterally, perhaps combined with saline. Since protein catabolism is severe, parenteral administration of protein hydrolysates becomes necessary. Another side effect is the rapid depletion of vitamin reserves. Therefore, multiple vitamins should be given, with special emphasis on the B complex group because of their importance in carbohydrate metabolism. Not infrequently in severe cases, these toxins, now increased in variety by faulty metabolism, attack the myocardium. This

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®1Lederle Laboratories Division, Pearl River, N. Y.
®2Chas. Pfizer & Co., Brooklyn, N. Y.
®3Parke, Davis & Co., Detroit, Mich.

mitigates a rather constant possibility of myocardial failure. When observed, digitalis in therapeutic rather than empirical dosage, is necessary.

Disorders of the Kidney

What of the kidney? A urinalysis always is mentioned in the literature as either a diagnostic or prognostic aid. It should read—several urinalyses. Great stress frequently is laid on laboratory findings on a single specimen as a basis for further tests or even to arrive at a diagnosis. I do not mean to speak disparagingly of laboratory tests, but rather to advocate a greater, broader use of them and an avoidance of too much dependency on single test results. A positive albumin reaction frequently is the criterion of kidney damage *per se* or of the need for further urine analysis. Remember that an albuminuria does not always mean nephron damage alone. It occurs also in such anoxic states as severe anemia, interference with renal blood supply, following severe exercise, and in cardiac failure. Of late, blood urea nitrogen measurements have been recommended as having great validity in measuring kidney function, especially in the case of chronic nephritis. Admittedly, chronic nephritis is the most common cause of an increase in blood urea nitrogen. But also bear in mind that it may be increased from other causes, such as: (1) Excessive catabolism from high fevers and in gastrointestinal hemorrhage with destruction and absorption of products, (2) in dehydration especially with increased salt loss, which thereby increases protein catabolism, and (3) congestive heart failure.

It remains that the state of the kidney must be watched and frequent urinalyses and blood urea nitrogen measurements made to serve as guides.

One of the first evidences of acute renal damage is an increase in specific gravity, presence of albumin, and of hyaline and fine granular casts in urine. These changes are the result of inflammation and increased permeability in the glomerula tufts and deterioration of tubular epithelium. What can be done when signs of acute renal pathology are seen? It may presage uremia and is a serious addition to already existing pathology. Fortunately the diuretic action of the previously mentioned glucose helps here and this action may be enhanced by using a 10% solution. Protein administration, either as food or as hydrolysates, should be reduced greatly since they will result in greater urea production and retention. Sulfonamides are contraindicated in in this condition, because of the danger of renal crystallization or blockage. Diuretics of the newer mercurial-protein combinations may be used with caution. Any nervous symptoms arising from cortical edema or congestion should be controlled with depressants.

Perhaps diarrhea, streaked with blood, may now have developed. Saline should now be replaced with Ringers solution, perhaps containing additional potassium. Intestinal astringents appear to have little effect, though the kaolin-pectin-bismuth compounds partially relieve the condition. Should recovery occur at this point. convalescense should be well supervised, with a gradual return to normal protein intake, and this only after the clinician is certain that no chronic kidney pathology remains. The vitamin administration should be continued for several weeks and the heart checked occasionally for signs of residual pathology. A hematinic might well be employed if anemia exists or persists.

Not all of our cases fare so well. Many leaving the hospital are rather battered from their recent bout. The most common persistent lesion is that of chronic interstitial nephritis. From this there is polydypsia and polyuria. The blood urea nitrogen remains somewhat above normal. Recurrent digestive disorders can be expected, as well as various degrees of skin pathology. The increased resistance to renal circulation results in a steady cardiac compensatory hypertrophy, dilatation, and perhaps with decreased efficiency. The lungs already scarred from their secondary bacterial invasion, receive poorer circulation and the typical cardiac cough and exercise dyspnea develop. This case requires a lifelong diet of low quantity, high quality protein. This may be accomplished by either a specifically prepared kidney diet, or a home diet of milk and milk products, cereals, eggs, limited meat, and adequate fat. The animal should be fed twice or three times daily in limited amounts. Periodic heart stimulation may be necessary and constant surveilance is essential. Continual efforts should be made to avoid the development of parasitism or of any infection to which the patient is now increasingly susceptible. With good care at home and the best of veterinary management, this dog may be expected to live a long and reasonably happy life.

Summary

This paper began with the assumption that the primary invading organism was a Leptospira. However, it should be realized that any

number of other pathogenic organisms could have been involved with much the same sequence of pathological events. Bearing in mind that it is internal pathology that produces the clinical symptoms upon which so much stress is laid, it follows that the clinical picture also would be much the same. It should be remembered that our cases are presented at varying stages in the train of events. It is for this reason that so many of these disease complexes are extremely difficult to differentiate. However, differentiate them we must, if we are to progress in our medical knowledge. Considering the complexity of the situation, it leaves little room for faulty thinking, failure to keep abreast of medical advances, or worst of all, to assume the validity of a diagnosis because we made it.

I hope that efforts to present this problem as a constantly moving chain of pathological events has been at least partially successful, and that pathology must be both anticipated as well as recognized if it is to be properly managed. A definite diagnosis always is desirable, but its definiteness must be considerate of both etiology and total pathology. The final name applied to the condition then becomes secondary. Severe symptoms of leptospirosis, with accompanying diarrhea, cough, secondary purulent discharges of the eyes and nose, and equally secondary impetigo seen in an eight-month-old puppy, which another veterinarian had vaccinated, might be misdiagnosed. Should the animal receive the usual course of serum and penicillin and die, it might be regarded as another distemper death. Should it survive, it would return home to be a constant spreader of the Leptospira organism to other animals and man. Obviously, then it is the etiology and the pathology with which one must be concerned most in the interest of proper disease management.

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Parasitic Diseases of Animals

(Continued from page 202)

diseases of obscure etiology in both man and domestic animals. Whereas, a few years ago, worms of other animals were considered innocent in man until proved guilty of offense, today it seems more reasonable to regard them all as guilty until their innocence is firmly established.

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Discussion Of Bat Rabies In Florida†

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THE recognition of rabies infection in insectivorous bats of the United States is a discovery of unusual interest which may have far reaching implications as a public health and economic problem. The existence of rabies infection in bats of this country was unknown until the isolation of rabies virus from a Florida yellow bat (*Dasypterus floridanus*) by members of the Florida State Board of Health in June 1953. Subsequently rabies virus was isolated by them from five Florida yellow bats and a Seminole bat (*Lasiurus seminola*), all of which were apparently normal. These two species are indigenous to the southern states.

Subsequent to the Florida discovery, rabies infection was found in a bat near Carlisle, Pennsylvania. This bat made an unprovoked attack upon a lady and bit her on the forearm during the latter part of September 1953. The bat was captured and died on the following day. Direct microscopic examination of the brain revealed Negri bodies, and the diagnosis of rabies was confined by rabbit inoculation tests. Unfortunately the bat carcass was destroyed before the genus and species could be determined, but there was general agreement among those who saw the bat that it was insectivorous[1].

Recognition of rabies infection in bats in Florida and Pennslyvania has revealed a probable reservoir and source of infection in a new group of wildlife whose feeding and living habits are entirely different from those of the animals heretofore known to harbor rabies virus in this country. It is too soon to state whether rabies in bats is localized to small areas of Florida and Pennsylvania, whether infection is spreading or receding, or whether it exists also in other parts of the United States. Certainly the possibility can not be ignored that bats may play a heretofore unrecognized role in the maintenance of wildlife rabies. Steps should be taken to obtain further information to determine the extent and importance of the problem. As an aid in understanding this new health problem, it seems desirable to present a brief review of bat rabies in Latin America, where the disease has existed for half a century. The designation "bat rabies" is used in the absence of better terminology for that form of rabies transmitted by bats, which usually occurs in man and in livestock as paralysis, but occurs in several forms in hemophagous and frugivorous bats of Latin America.

History and Distribution

The existence of rabies in bats was first recognized in Brazil during investigations of a fatal paralytic disease of cattle which had appeared there in epizootic form in 1908. In reviewing the early investigations which led to discovery, Pawan[2] recounted Carini's discovery, in 1911, of rabies virus in the brains of cattle which had died from this paralytic disease. He also recounted Rehaag's isolation of rabies virus in 1916 from the brain of a fruit-eating bat (*Phillostoma supercilliatum*) caught while biting cattle in the daytime. The inhabitants of the areas where Carini and Rehaag were investigating the paralytic disease in cattle had observed the peculiar behavior of bats in the area and were convinced that cattle bitten by them would die. Both Carini and Rehaag observed the abnormal behavior of the bats and suspected that the unusual fighting and fits were indications of disease. When cattle disease was shown to be rabies, dogs were implicated as being responsible for its spread, but drastic measures to destroy and control dogs had no limiting effect on the epizootic. With this information at hand, and following the isolation of rabies virus from the fruit-eating bat, Rehaag concluded that epizootics of paralytic cattle diseases in Brazil were caused by rabies transmitted by bats. Subsequently, bat rabies was observed in other South American countries.

Until 1929 all rabies suspected of being transmitted by bats had occurred only in animals, when suddenly there appeared, in southern Trinidad, an epidemic of ascending paralytic myelitis in human beings which Hurst and Pawan[3] found to be caused by virus of rabies. In 1936, Pawan[3] reported that his

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studies of rabies in Trinidad had revealed that vampire bats (*Desmodus rotundus*) transmitted rabies to both man and animals. During the past three decades bat rabies has been observed and reported in many other countries of Latin America[4,5,6].

Nature of the Disease

Bat rabies occurs in nature in man, in herbivorous animals, and in hemophagous and frugivorous bats. It has not been reported in dogs except in Venezuela[6] where bat rabies virus was isolated from them. The average incubation period in the *D. rotundus* is 25 days, but one bat showed probable symptoms on the third day following inoculation, then returned to normal until the seventh day when definite rabies symptoms appeared[7]. The longest known incubation period was in a vampire bat, infected in nature prior to capture, which remained infectious, without symptoms, for 5½ months[7].

Six forms of rabies were observed by Pawan[7] in vampire bats and were classified by him as follows:

1. The typical classical furious rabies in which fury is a prominent and predominating feature, followed by paralysis and death.

2. The typical paralytic form of rabies in which no state of fury or excitement can be observed, but in which paralysis terminates in death.

3. A furious form of rabies, in which the stage of fury is followed by recovery.

4. A furious form in which fury runs on directly to death with no intervening paralysis.

5. A form in which the bat is well, but dies suddenly and unexpectedly without any previous evidence of illness.

6. A subclinical or latent form of infection in which the bat continues to live without any apparent departure from normal.

In the classical form there is the usual incubation period, a prodromal or invasive stage, then a stage of excitement and lastly paralysis, ending in death. The prodromal stage of from 12 to 24 hours duration may be observed when the bat is restless, easily irritated, and there are muscular tremors, or when the bat may be apathetic and off feed. This stage is followed rapidly by excitement, fury, and various signs of deranged behavior. Paralysis may first appear as a paresis of one limb or may develop suddenly as a "stony" paralysis. It most commonly involves wing muscles and those controlling the bladder, producing incontinence of urine. The duration of paralysis is from one to five days and in its main features suggests the disease as seen in other animals and man.

The paralytic form of rabies usually is observed in cattle which have been infected by bats. It is characterized by an initial period of restlessness and excitement which soon is followed by a sudden onset of paralysis of the rear quarters. The common name for this disease in Mexico, derriengue, is derived from the Spanish word meaning a break in, or an injury to, the spine[4]. Affected animals ordinarily live for several days, during which time paralysis extends to the forequarters and neck. Salivation is noted early and is due to a difficulty in swallowing. Emaciation develops rapidly and the disease seems to be uniformly fatal. The sudden onset of posterior paralysis and the rapid extensive emaciation appears to be almost pathognomonic. The mortality during an epizootic is quite high. Johnson[4] reported losses of 20 to 50% in cattle in an area where derriengue was epizootic, and Schroeder[5] reported that single ranches in Honduras experienced losses of 40% in cattle during a four-month period.

Symptoms in human beings appear suddenly, usually three to four weeks following the bite of an infected bat. The first complaint is usually a burning or tingling sensation in the bitten limb and the affected limb may show paresis at the onset of the disease[8]. The patient is febrile and often has a headache. Following these prodromal symptoms, which may last from one to four days, muscular weakness progresses to complete paralysis of the affected limb. The trunk muscles and arms soon are involved, terminating in a fatal paralysis, usually about the seventh day after the onset of symptoms.

Epizootiology

The incidence of bat rabies is cyclical, and the disease in both man and animals occurs in epidemic form. Outbreaks usually are confined to relatively small areas and frequently are limited to valleys and arroyos which are inhabited by vampire bats. In Trinidad, however, the animal disease occurred in large numbers over the entire island at one time or another between 1925 and 1935, while the human disease remained confined to a few small areas[8]. The epizootics usually end in from one to two months, followed by appearance of sporadic cases for several months. Subsequently the disease may entirely disappear for one to two years before another outbreak. Seasonal distribution is quite constant, the in-

cidence peaking in January and February, with most of the cases in one year occurring between October and April[4]. This seasonal occurrence may be traced to the breeding habits of the bats. The first sign of an approaching epizootic is the existence of the disease in vampire bats as evidenced by an increase in the frequency of these bats biting men and animals. In epidemics, animal disease has invariably preceded the appearance of the disease in man.

The blood-lapping vampire bat is the proved transmitter of rabies and yet the disease has been recognized in four genera of frugivorous bats which were infected in nature. Although the nonhemophagous bats have not been considered important transmitters they have been found attacking animals when deranged with rabies and they do transmit the disease. Vampire bats are a constant threat as a source of rabies infection because of their normal living and feeding habits. They live in large colonies within easy flying range of the animals upon which they feed. Blood is obtained through a typical crater-like wound upon which several bats may feed at the same time, and they frequently return to the same host on succesive nights. The known habitat of the vampire is from the 30th parallel south latitude about the northern border of Uruguay, to the 28th parallel north latitude, about 30 miles north of El Fuerte, Sinola, Mexico. The many reports of rabies in widely separated parts of this area indicate that the disease presents a problem in all areas inhabited by the vampire bat.

Control

Control measures include destruction of bats know to transmit rabies virus and the immunization of susceptible animals. Bat populations can be limited effectively near villages or other thickly populated areas. Livestock may be protected, as is done in Mexico, by moving them 15 to 20 miles from areas where bat rabies is occurring[4].

An extensive immunization campaign was begun in Mexico in 1936, using a vaccine consisting of a 20% suspension of infected sheep brain in saline solution containing 0.5% formalin[4]. In 1951, Malaga-Alba[10] successfully used avianized vaccine prepared from the Flury strain to produce immunity against challenge with the derriengue virus. Schroeder[5] reported equally good protection in Honduras with the avianized Flury strain vaccine.

Summary

In the United States there has been little concern in the past that bat rabies would be encountered in this country because the known transmitter of the disease was confined, by climatic requirements, to warm areas some distance from our southern border. Since Johnson's[4] original report, Malaga-Alba[11] has found the disease in the Mexican states of Sonora, Chihuahua, and Tamaulipas which border on the United States. It is difficult to determine whether vampire bats are moving northward or if the bats have been present there for a number of years. The climatic requirements of vampire bats make it unlikely that this bat will present a problem in this country except perhaps along the southern fringe. Bats of one genus (Dasypterus), found to be infected in Florida, inhabit areas in Mexico which also are inhabited by vampire bats[13]. It seems quite probable that rabies could be passed and maintained among the different genera. It also is possible that bats could be infected by rabid carnivores and, in turn, could pass the infection on to other bats and animals.

The present information on rabies in bats of the United States is insufficient to permit more than a conjecture as to the public health and economic problems which now exist or which may develop in this country. Much additional study is needed on the prevalence of rabies infection in bats, the pathogenesis of the disease and the nature of the virus in them, and the transmission of rabies by insectivorous bats to other animals.

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EDITORIAL COMMENTS

Public Relations for Veterinarians

Brig. Gen. James A. McCallam, president of the A.V.M.A. has discussed the subject of public relations at various meetings during the past year. His paper, Veterinary Medicine and the Public, earned compliments at the Southern Veterinary Medical Association annual meeting in Atlanta, Ga., on Nov. 9-11, 1953.

President-elect Dr. A. H. Quin also has devoted time and energy to subjects pertaining to this important matter, having served on the A.V.M.A. Committee for Public Relations for several years. These two leaders have emphasized repeatedly that sound and productive relations with clients, allied professional groups, and the public at large is essential to healthy growth of veterinary medicine.

Public relations has its own technical skills and technics for accomplishing the mission of influencing favorable public opinion. It involves, briefly, interpreting the existence and objective of the profession for society. On reflection, personal opportunities of individuals become apparent. For those who come to know you, the profession will be judged by values you represent to them. Application of sound public relations technics not only assure personal successes, but the growth and prosperity of the profession is dependent on it.

Erysipelas Bacterin

A "newsgram" from the office of the A. V. M. A. advises of short-lived immunity in pigs vaccinated with the newly developed swine erysipelas bacterin. This note (full report to be published later) emphasized that while immunity was apparent in six pigs tested one month post-vaccination, no protection was afforded five months after such preventive treatment.

Erysipelas bacterin is prepared from stock cultures provided by the USDA. One of the requirements imposed by the Serum and Virus Control Division is that potency test applied to mice conform satisfactorily to stated standards. Mice are chosen, in this instance, as the preferred laboratory animal for tests of potency rather than pigs because of variability of susceptibility and response in the latter.

Results as shown by the Beltsville experiments, using weanling pigs, are not sufficiently conclusive to condem *per se* the efficacy of erysipelas bacterin. More tests on weanling pigs and older animals will be required before accurate evaluations can be made. It appears inconceivable that licenses to manufacture would be issued for production and sale of a product having as little practical value as an immunizing agent as suggested by the Beltsville experiment as interpreted and reported in this newsgram.

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Antabuse in Alcoholism

As a matter of general medical interest I was intrigued by an article in a medical publication about Antabuse in Alcoholism (World Health Organization uses the designation disulfiram as a synonym for antabuse). Antabuse, from the standopint of its chemical composition and therapy, is a highly purified form of tetraethylthiuram disulfide which is absorbed from the intestinal tract, oxidized in the liver, and execreted in the urine and feces. It acts by interfering with the normal body metabolism of alcohol. The drug is contraindicated if there is coronary artery disease, congestive heart failure, and those psychoses such as loss of memory, or difficulty in concentration. Though not particularly toxic, there are undesirable effects from over-dosing and idiosyncrasy. Therefore, the drug should never be given except under the supervision of a physician. Furthermore, there must be the strong desire on the part of the patient to overcome his desire for alcoholic beverages. The article further states that antabuse is an aid and not a substitute for psychotherapy.

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A recent poll of veterinary colleges indicates that there are 64 women students enrolled at the present time, 18 of whom are in the freshman year.—*Bul. W.V.M.A.*

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Bat Rabies in Florida

(Continued from page 208)

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American Animal Hospital Association, May 5-8, 1954

The 21st annual meeting of the A.A.H.A. is expected to draw record attendance. Visitors are promised an excellent program that will consider current professional problems of practitioners specializing in pet animal practice and a symposium on Hospital Services. The latter will include discussions pertaining to facility construction matters, records, employee-client relationships, legal and malpractice problems, and other items having to do with some business matters of concern to small animal hospital directors. The final session on Saturday morning, May 8, will be devoted to the subject of ophthalmology. Television demonstrations will be featured at this session.

An especially well-planned program of entertainment also is announced. A golf tournament on May 4, an evening at a famous night club, the president's dinner and dance, and sight-seeing trips have been arranged. Highlighting the latter is a tour of the United Nations headquarters, pictured below in an aerial view taken from a plane above the East River, showing (l. to r.) the Secretariat, Conference, and General Assembly buildings with the midtown Manhattan skyline in the background.

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Oxytocin, posterior pituitary hormone that controls aspects of parturition and lactation, has been demonstrated to increase production of milk and fat. Wisconsin studies (*Jour. Dairy Sci.*, 35:1117, 1953) revealed significant increases on administration of 10 international units of this hormone to a group of dairy cows over a 10-day period of observation. The ratio of milk and fat was unchanged.

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A New York City management survey specialist and an international police authority have recommended dogs for patrolmen for that city's parks. The report cited experiences in London where dogs were added to the police force in 1950. There handbag snatching, once the principal offense in Hyde Park, was reduced from 214 cases in 1948 to 18 in 1952. Credit for reduction in numbers of similar crimes is attributed to well-trained police dogs working with the foot patrolmen.

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The Texas and Southwestern Cattle Raisers Association estimated that 10,000 cases of hyperkeratosis occurred in west Texas during the first two months of 1953, and that 95% were fatal.

91st Annual Meeting of the A.V.M.A. Seattle, Washington, August 23-26, 1954

Those planning to attend the 1954 convention of the A.V.M.A. are urged to address the Housing Bureau, Seattle Hotel Association, 315 Seneca St., Seattle, Washington for accommodations rather than to selected hotels. A reservation form has been included in recent issues of the *Jour. Am. Vet. Med. Assn.* including that for April 1954.

Facilities in a single hotel are not available for the expected large attendance. Neither will all sessions be held in Olympic Hotel headquarters but in the adjacent theater building where seating and other accommodations are adequate.

Many visitors are expected to combine pleasure with the trip to this annual meeting and take advantage of the many vacation attractions of the Pacific Northwest.

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Trichinosis, serious, painful, and sometimes fatal disease in man, resulting from consumption of undercooked, infected pork will ultimately be eliminated as a public health problem as a result of legislation in most states requiring that garbage be cooked before it is fed to swine. Dr. Benjamin J. Schwartz, made the foregoing prediction during sessions of the second National Conference on Trichinosis in Chicago.

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Price and Hardy report (*Jour. Am. Vet. Med. Assn.*, 124:255 (Apr.), 1954) that on one occasion, in a series of transmission studies of bluetongue in sheep, subcutaneous and then intravenous injection of broth emulsions of *Culicoides variipennis* resulted in the production of clinical bluetongue in seven days. This finding substantiates some work in South Africa that suggests this species as insect vector of the bluetongue virus.

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"Dinner Bell" designates a WLS radio station feature scheduled for every other Tuesday afternoon at which time well-known veterinarians discuss livestock disease problems. It is made possible through the cooperation of the American Veterinary Medical Association, the Illinois State Veterinary Medical Association and the radio station management. The program has developed considerable active interest. A general discussion is announced in advance of each session. These broadcasts promise to be not only useful, by assisting growers, but an excellent public relation opportunity for the profession.

Campus View — University of Washington, Seattle

A view on the campus at the University of Washington, Seattle, one of the most beautiful college campuses in the United States. Buildings of historic Gothic architecture in a scenic setting of pine and fir trees are interlaced by streets and sidewalks plentifully landscaped with gardens and evergreens. The campus is located on the shores of famous Lake Washington.

Dr. James A. Farquharson Victim of Heart Ailment

Dr. James A. Farquharson died on Thursday night, March 11, 1954, at the home of his son, Dr. Bruce Farquharson, in Phoenix, Arizona. He had suffered several heart seizures

Dr. James A. Farquharson

during recent months; a previous one required hospitalization in a Kansas City hospital early in November 1953.

Doctor Farquharson came to this country from Scotland at an early age. He received his elementary education in Pueblo, Colorado, and his D.V.M. at Colorado State College, Ft. Collins, in 1921. The following year he took postgraduate training in anatomy at Iowa State College, Ames.

After a brief tour of practice in Colorado, Doctor Farquharson joined the staff of Colorado State College, School of Veterinary Medicine where, since 1934, he has been head of the Department of Veterinary Medicine, Surgery and Clinics. During this period he took further graduate work at the University of Minnesota, Mayo Foundation for Medical Education and Research.

Doctor Farquharson was perhaps best known for his demonstrations and lectures on surgery of both large and small animals. His reputation was nation-wide and he was in demand as surgeon and consultant. Valuable animals were moved from many distant points to Ft. Collins for his attention and treatment. His perfection of surgical technics and the clarity of presentation by well prepared motion pictures were such that he was in great demand as a lecturer at veterinary meetings, including the British Veterinary Association where his work recevied the highest praise.

In addition to his contributions to the advance of veterinary surgery, Doctor Farquharson earned renown as an an educator. He was admired and respected both for his skill as a surgeon and demonstrator, and for his sincere attitude toward educational matters including postgraduate training of practitioners. He served on the A.V.M.A. Education Committee for many years and as its president during the war years of 1944-1946.

Doctor Farquharson is survived by two sons, William Bruce, (Col '52), Pheonix, Arizona and James Jr., a student at Northwestern University, Evanston, Ill.

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Disinfectant Solutions

In a leaflet prepared by Livestock Conservation, Inc., there is given, in chart form, the following mixtures or solutions for disinfection following contamination from some animal diseases:

Disinfectant	Percent Solution	Mixture	Disease
Cresylic disinfectant (USDA Approved, marked on can)	4	1 cup to 2 gal. water	Brucellosis Hog chlorea Shipping fever Swine erysipelas Tuberculosis
Sal soda	10½	13½ oz. can to 1 gal. water	Foot-and-mouth disease Vesicular exanthema
Sodium carbonate (Soda ash)	4	1 lb. to 3 gal. water	Foot-and-mouth disease Vesicular exanthema
Sodium hydroxide (Lye)*	2	13½ oz. can to 5 gal. water	Foot-and-mouth disease Vesicular exanthema
Sodium hydroxide (Lye)*	5†	5 (13½ oz.) cans to 10 gal. water	Anthrax Blackleg

*WARNING: When using lye disinfectant of any strength, wear protective goggles, rubber gloves, and coveralls. Have a bottle of vinegar handy to use in case the lye solution comes in contact with any part of the body.

†When using a 5% lye solution, let it remain in the vehicle for 8 hours, then wash away with water before loading.

It is a good presentation to remember when advising livestock owners.

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Repeat breeders among cows may be early abortion cases, due to *vibrio fetus* infection.—*J. C. Osborn, D.V.M.*

PRINCIPLES OF ANIMAL HEALTH

R. R. DYKSTRA, D.V.M.
Manhattan, Kansas

Essential Vitamins for Cattle

Vitamins A and D are essential to mature ruminants. All others are either synthesized in the rumen or they are present in dry-lot rations. It is extremely doubtful therefore, that enrichment of good, practical rations with other vitamins is beneficial. Furthermore, vitamin D is not needed in extra amounts if there is sufficient direct exposure to sunlight, though animals kept indoors for prolonged periods need additional amounts. Vitamin A as such, occurs only in animal products such as those from the livers of salt water fish, milk, eggs, *et cetera*. It never has been found as such in plants. It occurs as an almost colorless solid, soluble in oils. Animals receiving no food other than that of vegetable origin get vitamin A from yellow pigments, the carotenoids, contained in green and yellow colored plant products which are converted in passage through the digestive tract into physiologically active vitamin A. Colostrum has a high vitamin A content.

This vitamin is essential in maintaining the epithelial cells of the skin, and in the mucosa of the respiratory and digestive tracts, as well as the reproductive tract. A cow weighing 1,000 lb. needs at least 50 mg. daily, and during late pregnancy as much as 100 mg. daily should be provided. Dark green color and the yellow of carrots and of yellow corn is evidence of the presence of carotene.

The usual clinical evidences of vitamin A deficiency are rough dry skin, night blindness, excessive lachrymation, and muscular incoordination. These symptoms are most likely to occur early in the spring months, especially if, because of drought, grasses during the preceding fall are fibrous and dry with an absence of the normal green color. The foregoing statement does not apply to calves before they ruminate, since during this stage of development they may suffer from both fat-soluble and water-soluble vitamin deficiency. In these young creatures, cough, diarrhea, incoordination and even spasms, as the result of nerve degeneration, may be observed. Since drought conditions in many parts of the nation have been rather general, it is well to guard against this deficiency.

New Antibiotic

New antibiotics continue to make their appearance. The latest, according to our information, is tetracycline, a product closely related to aureomycin (chlortetracycline) and terramycin (oxytetracycline). Tetracycline usually is marketed in the form of the hydrochloride as crystals. Compared with aureomycin and terramycin it is said to resemble closely these two in action. However, tetracycline in solution appears to be more stable than aureomycin, and when administered by way of the gastric tract, tetracycline is the least disturbing of the three products. The clinical effects are quite similar in all three antibiotics.

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Control of Scab in Sheep

Fiedler and DeToit in the *Onderstepoort Journal of Veterinary Research* (Vol. 26, no. 2, Nov. 53), report the result of their research on an evaluation of the acaricidal properties of aldrin and dieldrin for the control of sheep scab. They indicate that such chlorinated hydrocarbon insecticides as DDT and compounds related to it have a tendency to remain localized at the point of application so that they do not diffuse along the wool fibers into the new growth of wool. Toxaphene, chlordane, and especially BHC are readily diffusable. Dieldrin and aldrin were found to be even more diffusable than BHC. The conclusions of the authors are that dieldrin is an extremely potent and quick acting acaricide. Against *Psoroptes communis ovis*, it is able to control sheep scab with a single dipping at a concentration of 0.03% of the active ingredient. Its acaricidal action at this strength is comparable to that of gamma BHC at 0.02%. Aldrin, in wettable powder or emulsion form, at a concentration of 0.05% of the active ingredient, must be regarded as being insufficiently effective to eradicate scab by a single treatment.

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The U. S. Bureau of Agricultural and Industrial Chemistry isolated a microorganism from Japanese soil that is now used in the manufacture of 25% of the vitamin B_{12} used in stock feed.

POULTRY PRACTICE PROBLEMS

W. R. HINSHAW, D.V.M., Ph.D.
Frederick, Maryland

New References on Newcastle Disease

Two recent bulletins on Newcastle disease are recommended for your library. One of these, Facts About Newcastle Disease by Drs. B. S. Pomeroy and C. A. Brandly, Minnesota Agricultural Experiment Station Bulletin 419, June 1953, was sponsored by the North Central Regional Technical Committee for Newcastle Disease. The second, Newcastle Disease of Fowls, by Drs. E. P. Johnson, E. R. Doll, and W. A. Boney, Virginia Polytechnic Institute Agricultural Experiment Station Bulletin 464, Nov. 1953 was sponsored by the Southern Regional Technical Committee for Newcastle Disease. Both contain concise information on the disease with recommendations for prevention and control for the regions represented.

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Turkey Sinusitis

Infectious sinusitis of turkeys is no longer a mystery disease. Several investigators now have demonstrated that the etiological factor is a filtrable agent that can be cultivated in embryonating chicken eggs. It is the opinion of Groupé and Winn of the University of Connecticut (*Jour. Bact.*, 57:515, 1949) that it is caused by a filtrable agent belonging to the lymphogranuloma-psittacosis group of microorganisms (Chlamydozoaceae).

For many years before the exact cause was known, injection of 15% argyrol or 4% silver nitrate solution into the infected sinuses, following removal of the exudate, was an effective means of control of the sinusitis type of the disease. These treatments were of no value in cases that also had air sac or lung involvement. In 1949, Hitchner (*Poultry Sci.*, 28:627) reported the successful use of streptomycin as a curative agent. Since then several reports on this and other antibiotics have appeared in the literature.

The first successful attempt to control the disease by feeding antibiotics has been published recently by Grumbles and Boney (*Jour. Am. Vet. Med. Assn.*, 119:384 [Nov.], 1951). They fed chloromycetin® and terramycin at various levels in an all-mash ration to turkeys artificially infected. Chloromycetin at levels of 0.25 and 0.5% for eight to 12 days successfully cured a high percentage of the sick birds in seven to 14 days. At a level of 0.1%, the drug was ineffective. Terramycin at levels as high as 0.5% fed for seven days was ineffective. The most significant factor in these trials was the response of the lower respiratory infections which has not been noted by treatment with any other drug.

®Parke, Davis & Co., Detroit, Mich.

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No entirely satisfactory treatment for salmonellosis has been found. Sulfonamides, if used according to recommendations given for each individual kind, may be of value in reducing losses if treatment is started early in an outbreak. Several of the sulfonamides have proven toxic if given in too large dosage. It is important to remember that carriers are not eliminated by treatment so flocks which have recovered should not be used as breeders.

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Oleander (*Nerium oleander*) is poisonous for both man and animals and produces symptoms similar to digitalis poisoning. Although no authentic cases of oleander poisioning in poultry are known, poisoning has been artificially produced in turkeys. McNeal and Denny (unpublished data, University of California, 1939) killed five of six four-week-old poults by inserting leaves of oleander sprouts into their crops. The same poults refused to eat the leaves when offered as greens. The poults died within 24 hours after being force-fed and, on autopsy, revealed a marked hemorrhagic enteritis.

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U. S. Department of Agriculture, Circular 932, 1953, An Analysis of Commercial Frozen Egg Products is a report on research to determine useful, rapid, and economical methods of evaluating the quality of commercially produced frozen eggs moving in marketing channels. Methods of obtaining samples and making examinations of these are described. An *E. coli* count designed to obtain an approximate numerical estimate, by a modified counting technic, was used instead of the usual dilution procedure. The report should be of interest to those who are concerned with veterinary public health.

CLINICAL REPORTS

Toxoplasmosis in a Cat

H. C. SMITH, D.V.M., Sioux City, Iowa

The case of toxoplasmosis here reported should be of interest to small animal practitioners and veterinarians engaged in public health work, because it is the first instance, to our knowledge, in which the protozoa has been diagnosed by tissue sections in the spleen of a cat.

This disease has been reported as occurring in man, dogs, cats, swine, sheep, cattle, monkeys, chinchillas, rabbits, pigeons, wild birds, kangaroos, and many rodents.

Toxoplasma from one species of animal may infect a wide variety of animals. The exact mode of natural infection is not known. The disease has resulted from the ingestion of infected carcasses and this may be an important factor in its transmission in the animal kingdom[8].

Experimentally it may be reproduced by injection of infected material intravenously or intraperitoneally into the laboratory animals.

An accurate diagnosis is made only in the laboratory on finding the protozoa. On postmortem examination it may be mistaken for Listeria and Sarcosporidia. In animals the disease may be a chronic, latent condition.

Sabin and Olitsky[11] first proved the existence of the protozoa in North America when they isolated it from a guinea pig.

Olafson and Monlux[6] were the first to report this disease in a cat. The case was a one-year-old spayed female. It had no appetite for three days, temperature ranged from 103 to 105 F., loss of body weight was rapid. Treatments with enteritis antiserum and sulfonamides were of no value. Postmortem examination revealed enlarged mesenteric lymph glands, small ulcers in the mucosa of the intestines. The lungs revealed many multiple, firm, white nodules resembling tumor tissue with central necrosis. Toxoplasma could be demonstrated from the bronchi and epitheleal cells of the lungs.

Several investigators have discovered the condition in dogs[11,9,10]. Both intra- and extracellular parasites were found in the liver and spleen lesions marked by cellular degeneration. Olfson and Monlux[6] described the dog as manifesting labored breathing, loss of appetite and high temperatures. The lungs were congested and edematous and contained foci of necrosis. Ulcers in the stomach contained the protozoa.

Moulton and Linto[7] state that their case in a dog was of an acute nature. The animal became progressively emaciated, with persistant diarrhea and heavy breathing. The pathology was an exudate in the pleural and abdominal cavities. The spleen was enlarged. The intestinal mucosa contained ulcers.

Postmortem lesions of toxoplasmosis in a cat. There were shot-like areas in the lungs, liver, and spleen with central necrosis.

Chamberlain *et al.*[1] made inoculations of Toxoplasma from cases in man, dog, and swine. These were injected into five bitches in various stages of pregnancy. Premature abortions occurred in all five animals. The Toxoplasma protozoa were isolated from the pups and the placentae and from the milk and mammary glands of the dams.

Manwell *et al.*[5] transmitted the disease in canaries by ingestion of infected material but failed to reproduce the disease in other fowl by direct contact.

Olafson and Monlux[6] describe the disease in sheep as manifesting no gross lesions. The symptoms were labored breathing, normal temperatures, nasal discharge, muscle rigidity, and nervousness.

Sanger *et al.*[12] isolated the parasite by injecting the milk from infected cattle intra-

peritoneally into Swiss mice. The liver of a calf, born from an infected cow, harbored the organism. Calves and two-year-old heifers inoculated by various routes developed the disease. They exhibited a respiratory and central nervous system disease. In calves, the infection may be acquired *in utero*.

The disease in cattle is characterized by coughing, sneezing, rapid breathing, frothing at the mouth, trembling, head shaking, loss of appetite, and prostration just before death.

Hatchel *et al.*[4] describe the pathological findings in a kangaroo. The myocardium contained necrotic foci. The mucosa of the stomach was edematous. The lungs were speckled with grey-white nodules.

Farrell *et al.*[2,3] isolated the parasite from swine. They found that the disease occurs in pigs from one day to one year of age. Deaths were greater in the younger group of pigs. The lesions were pneumonia, lymphadenitis, ulcerative and fibrinonecrotic colitis, and hepatitis . There was considerable exudate in the pleural and abdominal cavities.

Lesions of toxoplasmosis in a duck.

Keagy, Hilan F. C.[5] found that the death rate was high in chinchillas. Affected animals had labored breathing, loss of equilibrium, and nasal discharge. Examination of carcasses showed hemorrhagic lungs. The spleens were enlarged, as were the mesenteric lymph glands. The protozoa was isolated from the lungs, lymph glands, and brains.

According to Walfson[1,3] the parasites can be identified in chicken, duck, and canary erythrocytes. The parasites could be grown in 14-day embryos of chickens and ducks and in tissue culture media. They could not be grown in artificial media unless it contained living tissue.

Case History

Tissues of a kitten were submitted to this laboratory for bacterial examination. They were sent by Dr. J. O. Buck of Sioux Falls, South Dakota and consisted of pieces of lung, kidney, and spleen.

Both aerobic and anaerobic cultures were made, but after 48 hours of incubation no bacteria grew. The kitten had been owned only seven days when it became sick.

Symptoms

The temperature was 104 F. There was no coughing or discharge from natural openings. Moist rales were heard over the pleural cavity when examined with a stethoscope. The kitten refused food and was beginning to become dehydrated and lose body weight.

Post Mortem Findings

There were shot-like areas in the lungs, liver, and spleen with central necrosis. There was considerable amounts of yellowish fluid in the heart sac and in the pleural and abdominal cavities. The digestive tract was empty, but appeared normal.

Microscopic sections of the spleen tissues revealed the presence of Toxoplasma parasites.

Comment

A diagnosis of toxoplasmosis is difficult. Almost any warm-blooded animal appears to be susceptible to infection. Any animal that shows unusual areas of necrosis in the glandular organs, along with exudates in the pleural and abdominal cavities on autopsy, should be examined for toxoplasmosis. Since the exudates usually are loaded with the parasites, they make an excellent medium for inoculation into mice and guinea pigs as test animals.

Proper identification of the protozoa is the only satisfactory way of making an accurate diagnosis.

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Field Observations on Granular Vaginitis in Cattle

JOHN B. HERRICK*, D.V.M., Ames, Iowa

Granular vaginitis is a specific, contagious inflammation of the reproductive tract of cows particularly affecting the vulvar and vaginal mucous membranes. This disease affects young heifers much more severely than older cows. A periodicity in this condition has been observed; symptoms reach a peak of severity and gradually subside in the course of a few months. Granular vaginitis is a common disease where natural breeding service is used. However, it is seldom observed where artificial insemination is used exclusively. It is of utmost importance not only because it affects the conception rate of the breeding herd, but also because it may mask symptoms of other diseases such as trichomoniasis.

In routine sterility work on naturally serviced herds, vaginitis may be observed reducing the breeding efficiency by as much as 50%, especially in heifers. In infected herds as many as four or five services per conception may be necessary [1,2].

In all probability, the infection is spread by the breeding process, but there is no reason to doubt that it can be spread by other means also. If the penis of the herd bull is examined carefully and thoroughly with the aid of a pudendal block, lesions on the mucous membrane similar to those found on the vaginal mucous membrane will be observed. Therefore, treatment of the sire is necessary in any control measures. As far as is known, the disease is not spread by artificial insemination.

Sexual rest and topical treatment of the involved portion of the reproductive tract greatly aid in improving the conception rate in infected herds. In one herd where the disease was evident, 60 females received two insufflations, four days apart, of 5 gm. kaolin-silver picrate compound**. All breeding was suspended for 30 days. The two males in the herd received suitable local treatment with antibiotic compounds. Two weeks after the last treatment, symptoms had subsided; this was especially noticeable in the heifers. When breeding was resumed, efficiency increased from 48 to 71%, based on 60 to 90 days non-return for service.

Another dairy herd consisting of 40 breeding animals, following the removal of the bull, was treated with kaolin-silver picrate compound twice, with an interval of four days between treatments. Artificial insemination was instituted later. The breeding efficiency increased from 52 to 68%, based on 60 to 90 days non-return for service. The lesions of vaginitis were not visible on the heifers following treatment. However, some lesions were visible on a few of the older cows.

An increased abortion rate is not common when vaginitis alone exists in a herd. An increase in the number of abortions suggests the presence of brucellosis, trichomoniasis, leptospirosis, or some other infection.

Uncomplicated granular vaginitis, however, obviously does affect conception rate. Topical treatment and sexual rest aid in controlling the disease and increasing breeding efficiency. Lugol's solution and other compounds have been used in the treatment of granular vaginitis with apparent success when breeding operations have been suspended or artificial insemination employed. Kaolin-silver picrate compound has the advantage of easy application by an insufflator. The only precaution necessary is disinfection of the insufflator tip following treatment of each individual.

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Severe Gastroenteritis In a Leopard

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On August 22, 1953, I was called to the city zoo to examine a pair of leopards, one of which had vomited and had a very loose and slightly bloody stool. This animal, a one-year-old male, had not eaten the previous day and had passed a rather mucoid bloody stool during the night. After the attendants arrived in the morning, the cat vomited a frothy substance, and later bile. Being a young and rather tractable animal, I had no difficulty takingits temperature, which was over 105 F.

My first though was the possibility of infectious enteritis (feline panleucopenia) and as I had not enough serum on hand, I wired for an additional supply. I prescribed the im-

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**Compound Powder Picragol, supplied by Wyeth Laboratories, Philadelphia, Pa.

mediate administration of one tablespoonful kaopectate and neomycin, plus two teaspoonfuls of syrup of aureomycin every three hours The temperature remained high the following morning and medication was continued. When the serum arrived that afternoon, 100 cc. was given to the affected animal and a prophylactic dose of 50 cc. to its cagemate. On the third morning there appeared to be some improvement and an additional 50 cc. serum was given. The meat from a fresh-killed rabbit was removed from the bone and fed to the leopard, which consumed it immediately.

The temperature returned to normal on the fourth day, and medication was continued with the addition of a vitamin B complex preparation. After 24 hours all treatment except the vitamins was discontinued. After ten days complete recorvery was evident and the cat was returned to its exhibition cage.

No laboratory studies were attempted and therefore no confirmation of the clinical diagnosis could be made. There has been no appearance of any similar condition in any of the other zoo cats to date. We have, inoculated our Bengal tiger, one of our most valuable animals, and we plan to inoculate the remainder of our young felines.

I will appreciate any information relative to this condition from veterinarians who have experienced similar disease manifestations in zoo animals.

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Genital Infection of Bulls

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A new disease entity of bulls, maintained at cooperatives for artificial insemination, is reported that has concerned officials of these organizations and attending veterinarians.

A good working bull suddenly loses libido although appetite and general appearance is normal. When observed at the time semen is collected, the penis appears normal at first, then red spots the size of a pinhead appear on the surface of the organ. These become extremely inflamed and often some rupture and ooze serum or blood from fissures. This development is accompanied, usually, by thickening of the spermatic cord. Swelling of the cord is prominent near the inguinal ring.

Affected animals were sampled with a pipette used for collecting specimens for examination for trichomoniasis. Smegma collected, and examined bacteriologically, revealed the infection as an alpha hemolytic streptococcus of the *Strep. viridans* group. During the last half of 1953, we found seven bulls infected. Six of seven infected bulls, at one artificial insemination establishment, were housed in adjacent quarters, while one was stabled in a distant wing of the barn. The only common place that these bulls came in contact was at the collecting ring.

Treatment

Infected bulls were treated with 6,000,000 units of penicillin in oil each day for four days with good results. Bulls that normally worked well before infection are restored to usefulness.

Efforts to improve sanitary conditions in the collecting ring by covering the dummy with a sterile shroud so far have failed because no material yet has been found which satisfactorily will stand the abuse given by heavy animals.

All bulls were sampled after treatment at intervals of two to five weeks. All responded to the first treatment series except two. These were retreaed with 20cc. dihydrocillin® daily for four days. These animals were negative after the second series of treatments.

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Toxoplasmosis in a Cat

(Continued from page 215)

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We are indebted to Dr. W. S. Monlux, Iowa State College, for his assistance.

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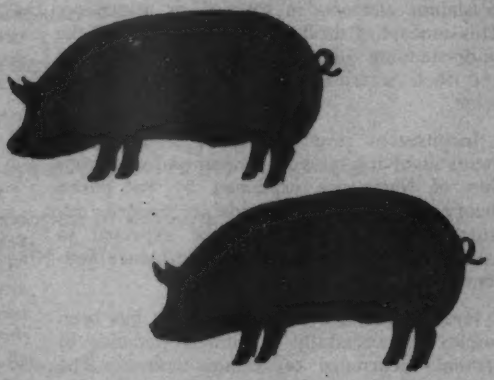

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ABSTRACTS OF LITERATURE

Colloidal Theory of the Cause of Urinary Calculi

Etiology of urinary calculi in sheep and cattle so far has evaded extensive research efforts. Consistent results have not been obtained though theories concerning vitamin A deficiency, excessive intake of minerals, and decreased consumption of water have relative value. A new theory concerning the significant role of urinary colloid gives promise of a better understanding of the reasons these stones develop[5].

Chemical analysis of kidney and bladder stones has revealed that they are concentrations of crystalloids bound together by, and incorporated in, a colloidal matrix. Ultramicroscopic studies of urine of sheep and cattle reveal tiny particles which are in constant, irregular movement (Brownian motion).

These particles are colloids. A dried film of urine from healthy, normal sheep, when studied under the ultramicroscope, reveals crystals surrounded by colloids. Study of urine from animals suffering from urinary calculi reveals crystals in aggregate without surrounding colloids. These observations suggest the protective role of urinary colloids, which, by movement and other means, tend to maintain crystalline elements in solution or dispersion. This concept of urolithiasis is supported by the understanding of the properties of colloids and their interplay with electrolytes of the urine.

Inconsistent results obtained from experiments involving mineral feeding and consumption of water are explained by the above theory. If sufficient colloids are present, nutritional factors would not be expected to cause calculi where they might influence concentration of urinary colloids.

Hyaluronidase, a dispersing agent, has been employed successfully in human medicine to prevent recurrence of kidney stones. The author reports significant results suggesting the use of hyaluronidase as a preventive of urolithiasis in livestock. More research on the matter must be done before definite conclusions can be drawn.

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Rumenitis Due to Rapid Change of Feeds

Liver abscesses in beef cattle are secondary to rumenitis, according to research studies in Colorado.[7] Rumenitis, caused by excessive fermentation may be brought on by changing from roughage to grain too rapidly.

Late tests indicate that digestive disturbances were common in 57 cattle changed to a full feed of concentrates (grain and supplement) in 12 days.

At slaughter, these animals had approximately three times the inflammatory processes of the rumen lining than those brought to full feed in 30 days. Differences in gains over a 133-day feeding period were small for the two groups. Supplementary calcium carbonate did not prevent injury.

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Diseases of the Retina

Steel, McGovern, and Keep report 20 cases of clinically delectable retinopathy in mongrels and common breeds of dogs[8]. Retinopathy was characterized by defective vision, gradual loss of retinol transparency and accumulation of pigment masses. Pathologically, it was characterized by pigment alterations and edema. The authors observed clinical association between the disease and the attack of distemper-like illness, but are unable to reach definite conclusions as to etiology.

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Parasites of the Orbit

Several ocular parasites of domestic animals are known in the United States. C. W. Schwabe summarizes these in a recent article[9]. "Those that are of actual or potential import are *Oxyspirura mansoni* in poultry; *Thelazia californiensis* in dogs, sheep, and deer: *Thelzia rhodesia* in cattle; and screwworm larvae in all species." Diagnosis is generally made by observation, and treatment consists, in almost all cases, of manual removal followed by routine treatment for inflammation.

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